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PNEUMONIA

THE
NATURAL HISTORY AND RELATIONS
OF
PNEUMONIA

ITS CAUSES, FORMS, AND TREATMENT

A CLINICAL STUDY

BY

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PREFACE

SINCE the publication of the first edition of this book in 1876 many subjects of interest and practical importance have arisen in connection with pneumonia, and its literature is now very extensive. It has been necessary, therefore, not only carefully to revise the original work, but also to add much new matter.

The views held in regard to the pathology of the disease in the first edition are here, for the most part, retained. But in the interval much has been done by many workers at home and abroad to establish, on a firmer basis, opinions which, fourteen years ago, were mainly hypothetical. Of the details of such investigations—and particularly of inquiries bearing upon etiology, epidemics, infection, and the influence of micro-organisms,—the authors have endeavoured to give an unbiassed account without anticipating that ultimate judgment for which, in their opinion, the material is as yet insufficient.

Information has been sought from many quarters. It will not be disputed that the names of Grisolle, Jürgensen, and Wilson Fox stand pre-eminent in their respective countries as historians of pneumonia, and in the following pages they are often referred to. Besides these the chief authorities consulted will be found enumerated in a separate Index.

For the clinical illustrations, which form a prominent feature in the present volume, the authors desire to express their obligations to the hospitals they have respectively the honour of serving, to the Middlesex Hospital, the Westminster Hospital, and the Hospital for Sick Children. They have also to repeat the thanks rendered in the former edition to the Medical Staff of St. George's Hospital for information derived from the clinical and post-mortem records of that Institution.

The Collective Investigation Committee of the British Medical Association, in 1884, gathered particulars of more than a thousand examples of pneumonia occurring in various parts of the United Kingdom. By request of this Committee the authors drew up a report based upon the material thus collected, and they have here made free use of this source of information.

Thanks are especially due to Dr. R. G. HEBB, Assistant Physician and Pathologist of the Westminster

Hospital, for several excellent microscopic preparations, and to Dr. H. W. SYERS and Dr. W. A. WILLS, Medical Registrars in succession at the same hospital, for clinical notes of cases. The authors are under similar obligation to the Middlesex Hospital, and especially to Mr. LEOPOLD HUDSON, Curator and Pathologist (now Surgical Registrar), and to Messrs. E. E. LEWIS and MONTAGUE TENCH, House Physicians during 1888 and 1889. At the Hospital for Sick Children, Dr. A. F. VOELCKER, Medical Registrar and Pathologist, has kindly supplied some statistical tables and clinical notes.

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PART I

CLINICAL HISTORY AND MORBID ANATOMY

CHAPTER I

PNEUMONIA IN HISTORY

Early accounts of the disease—Its appearance in certain historical epidemics—Association with the influenzas of this country—Various forms of pneumonia in the past, and their treatment—Growth of knowledge of the disease due to the progress of anatomy and the discovery of auscultation—Two types of pneumonia recognisable.

THE history of Pneumonia within the present century reflects the history of medical practice in general during a period of singular restlessness and change. By tracing successive doctrines in reference to this single affection from the time when the genius of Laennec first made the study of it possible, we may follow without break, up to the views of to-day, the several principles and modes of thought which have governed pathology in the same interval. Pneumonia is a representative disease. Discussions as to the nature and results of inflammation have chosen it for their chief illustration, and the effect of antiphlogistic treatment has been condemned or approved upon its evidence. When depletion was most in vogue it was to the lung in inflammation that its methods were most relentlessly applied. When the wisdom of bloodletting began to be questioned, it was resolved to test its efficacy by appealing to the results obtained in pneumonia ; and, coming to later times, when disease was first recognised as consisting in an orderly succession of phenomena, it was again with pneumonia that the crucial experiment was made of leaving inflammation to its own course.

Thus the history of this disease, if only we can faithfully depict it, will be found to comprise something more than the bare enumeration of a certain set of symptoms, or the barren record of a succession of remedies, vaunted at one moment and abandoned the next. It will be an epitome of the changeful doctrines of pathology in past generations ; of the facts once relied on to sanction a system of therapeutics far more peremptory than ours ; and of the arguments which have since prevailed to modify, or, more truly, to revolutionise the old canons of treatment in acute disease.

And, while thus interesting and important as the type and pattern of inflammatory affections, pneumonia occupies a place of its own, distinct both from strictly local diseases and from those which affect the system generally. Differing from either, it has something in common with both. Besides the pulmonary lesion there are certain general phenomena, involving other organs, with a train of symptoms so orderly as to time and succession as to resemble rather the course of a specific fever than that of a mere local inflammation.

Yet most of all is the study of this disease to be commended for the promise that attends it. There is a hope and purpose here, keener and brighter than in those organic affections where the utmost effort of art serves only to lengthen out a life which it can hardly render tolerable. Pneumonia, like fever, may attack those who are in the full vigour of life. It is of the deepest concern, therefore, to the public welfare to be resolved how to meet it. The issue in life or death depends, we know not as yet how much, upon the sagacity and discernment of the physician. Within our own time the mortality has been lessened by the independent exercise of these qualities with a courage that has not hesitated to break with the old traditions. Small as is the sacrifice to pneumonia to-day in comparison with the past, it may be yet further diminished by perseverance in the same patient observation of nature which has already been richly rewarded.

The early history of pneumonia is very obscure. Accounts given by ancient writers are not, in fact, sufficiently precise to make it certain that successive authors are describing the same disease. All that can be said is that a condition which, after the nomenclature of Hippocrates, went by the name of *peripneumonia* occupies a conspicuous place amongst the maladies of old. It is clear that the ancient physicians failed to distinguish between pneumonia and acute pleurisy. Hippocrates, obviously confusing the two, describes the latter as a disease of rapid fatality, exhibiting variously coloured sputa. Even our own Sydenham speaks of the two affections as forming one malady ; while Cullen, writing only a century ago, comprehends under the title pneumonia the whole of the inflammations affecting either the viscera of the thorax or the membrane lining the internal surface of that cavity. 'Neither do our diagnostics,' says he, 'serve to ascertain exactly the seat of the disease, nor does the difference in the seat exhibit any considerable variation in the state of the symptoms, or lead to any difference in the method of treatment.' As lately, indeed, as 1792, Jean P. Frank, in contradiction of some indistinct affirmations of Valsalva and Morgagni, maintained that pleurisy and pneumonia must be studied under the common name of pleuro-pneumonia.

While thus loosely described, it is significant that in the works of Huxham and Hoffman, over two hundred years ago, the term 'pneumonic fever' is adopted, indicating, as Grisolle has pointed out, the belief of the authors, even in that early day, that a special fever, rather than the local condition accompanying it, is the primary matter.

In spite of its imperfect definition, it was yet thought necessary to distinguish the real disease from its supposed counterfeits. 'True,' 'acute,' 'legitimate,' on the one hand; 'false,' 'spurious,' 'bastard,' 'illegitimate,' on the other, are expressions that have been in use for nearly two centuries.

Traces of the disease we are to describe may be found scattered about the medical history of Europe from the earliest times. In many of the great epidemics of which accounts are preserved the stress of the sickness has fallen chiefly upon the lungs, and contemporary writers have described these organs as 'inflamed,' and named the disorder accordingly. Thus the plague at Athens, 430 years before Christ, has been represented as a gangrenous pneumonia. The account of Thucydides,[1] however, seems to apply rather to a disease in some respects resembling typhoid fever, yet with special features of its own differing from all known epidemics. The extent and the manner of the lung implication it is very difficult to determine. The chief danger of the disease, and its most frequent termination, was by 'ulceration and excessive diarrhœa, by which the patients were afterwards carried off through mere weakness.'

Epidemics much later in time than this are involved in much the same obscurity. Take, for instance, the Black Death of the fourteenth century, which devastated Asia as well as Europe, and destroyed, as was said, two-thirds of the people, besides beasts, birds, and even fishes.[1] The leading symptoms of this pest, as described in a manuscript found in the library of St. Pierre at Lyons, were—cough, bloody spitting, diarrhœa, and vomiting, 'together with buboes, anthrax, and petechiæ.' The learned monk, whose curious parchment, written in verse, was accidentally brought to light, recommends as preventives living in a pure air, following a very moderate rule of life, avoiding cold and damp and fœtid emanations, and 'keeping oneself pure as at the day of baptism;' advice of such enlightenment as needs some acquaintance with the practical medicine of the fourteenth century fully to appreciate.

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[1] Book ii. chap. xlix.

Upon such meagre information, and in the absence of any trustworthy post-mortem description, we can say no more than that these ancient epidemics resembled generally certain prevalent fevers of later date, in which symptoms of lung mischief occupy a prominent place, and death discloses engorgement or consolidation of those organs. The earlier part of the sixteenth century was remarkable for a series of epidemics apt, under certain conditions, to assume this character. In connection with the general scarcity and famine which marked the revival of learning, a pestilence of this kind extended over Italy, France, Hungary, and Spain. It is described as a maculated fever, and the discussions of the day had reference to its relationship to the true plague.

Later, in 1557, while an epidemic resembling typhus was prevailing in France, an offshoot from it arose in Belgium which would seem not unfitly described as epidemic pneumonia. Commencing at the end of September, it was preceded by a violent and very cold north wind, whereupon 'catarrhal affections commenced, followed by vehement cough and fever, pain in the side, and difficult respiration ; bloody expectoration occurred on the third day, and death on the fifth to the eighth, unless bleeding was performed on the first or second day.' Practised later, venesection was held to be useless.

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[1] Ozanam, vol. iv. p. 76 *et seq.* The statements that follow in reference to the old epidemics are chiefly from the same authority.

A similar sickness occurred early in the seventeenth century. Dyspnœa, cough (dry at first but afterwards viscous, and, later, rust-coloured or bloody), sharp pleuritic pains at the onset, and at the end delirium. The disease, so similar in these respects to our pneumonia, was supposed to be decidedly contagious. The post-mortem appearances, given in vague terms, may be variously interpreted by the modern pathologist :—'Fœtid water' in the pericardium ; 'a pituitous substance,' bloody and purulent, in the lungs.

An epidemic, still better entitled to be called pneumonia, attacked the garrison of Philippsburg in 1688. It was attributed to long-continued cold north winds, along with the privations and exposure of camp life. At first, as the account runs, the sufferers were easily cured by bleeding, but soon the malady assumed a markedly epidemic character, and, aided by measles and small-pox, made great ravages, especially among the more dissolute of the soldiers. Its symptoms, shortly described, were 'stitch' and chest oppression, terminating, in the fatal cases, in delirium, or convulsions, or diarrhœa, or in all three. The usual duration in these instances was from seven to nine days. There is the exceptional interest attaching to this particular epidemic that the post-mortem condition is given in some detail. The lungs are described as 'actively inflamed and hepatised, in many parts purulent, the chest and pericardium filled with bloody serum, and polypi in the right auricle of the heart.' From this last appearance, and from its high mortality, the epidemic got the name of malignant 'polypous peripneumonia.' It need hardly be said that the description, so far as it goes, nearly resembles the pneumonia of to-day, polypi signifying ante-mortem clot (probably the earliest historical reference to it) and the contents of the pericardium pericarditis.

Coming to the eighteenth century, we encounter a contagious and generally fatal pulmonary epidemic raging in Rome. It was characterised by jaundice; and deserves mention in illustration of the fact that our fathers were not slow to discover the failure of their favourite remedy, phlebotomy. Two epidemics—a sthenic and an asthenic, as we might say—seem to have succeeded each other. In the first, which was 'of inflammatory character,' bleeding was beneficial. In the second (in 1713) 'gangrene' was apt to supervene both in the lungs and other organs; and now the operation of bleeding, useful before, was believed to be fatal. Of the earlier epidemic cold weather was thought to be the exciting cause. It disappeared with the advent of summer, and was again provoked, as was supposed, by a cold north wind and rains, under the form of a 'malignant pleurisy.'

It would be profitless, with the scanty materials at hand, to allude to other epidemics supposed to be pneumonic. In circumstances and phenomena they have much in common. Associated almost always with inclement weather, especially with prolonged and cold north winds, their history included symptoms (as in the epidemic of 1750 described by Roulin) nearly resembling the influenzas of the present century. Great prostration and limb pains, sometimes with and sometimes without lateral 'stitch,' concur with rust-coloured spitting and other signs of proper pneumonia; 'the cheek of the affected side being commonly covered with a dark red patch,' and the malady ending by critical sweats with cessation of fever from the fifth to the ninth day. Such was the epidemic of Flanders in 1756, and of Eplechin in 1776, where the likeness to pneumonia is very striking. Diarrhœa, bilious vomiting, and jaundice are other symptoms of frequent occurrence. And, as with influenza, so here, bleeding, in some instances resorted to as the only remedy, was in most regarded as absolutely fatal. Of 95 bled at Berne in 1762, 85 died, while only 10 died of 77 who were not bled.

As has been said, it is seldom up to the end of the eighteenth century that any further light is obtained from post-mortem inspection. Such scanty observations as we possess are open to various interpretation and remark. Thus while the lungs are often described as 'gangrenous,' the well-marked features of gangrene, and especially its fœtor, which would hardly have escaped record, do not appear in the life history. Some of the descriptions would serve well enough for grey pneumonia, as where the lung is described as covered with 'an ash-coloured membrane,' and its substance 'as falling away into putrilage.' It is to be remarked, however, that the morbid condition, whatever it be, involves the lungs wholly; it is never a single lung which is inflamed, or gangrenous, or sphacelated. Often, too, 'gangrene' affects other organs, and especially the liver, while not seldom ulceration or 'gangrenous aphthæ' are found in the intestines.

The presence of recent pleurisy, which with us is regarded as a valuable indication of the character of the disease, finds little mention in these earlier records. The reason no doubt is that the visceral pleura was not recognised in those days as distinct from the organ it envelopes. Pleurisy applied to the costal pleura only, and the word is mostly used to denote serous effusion into the pleural cavity.

In many instances, however, it must be confessed, the language used is altogether ambiguous, as where the heart is 'withered' and diminished in volume, or the pericardium contains 'fœtid water.' Pathological nomenclature, even with us, is shifting and fanciful; the same phrases carry one sense to-day and another to-morrow. But at the present time we have the advantage, that the artist lends his aid to render description durable by lifting it above verbal changes.

It will occur to every reader to contrast these desolating epidemics of so-called pneumonia with the historical influenzas of our own country. While the former seemed to threaten almost the extermination of mankind, nothing is more remarkable in connection with the influenzas than the insignificance of their mortality. Although whole populations were stricken, and the symptoms (including high fever and considerable bodily and mental prostration) were apparently grave, death occurred only as an accident.[1] 'I know not,' says Heberden, speaking of such an epidemic a hundred years ago, 'who could properly be said to die of it.' Nevertheless it seems highly probable that the very same epidemic which in Europe was exhibiting pneumonic symptoms, and obtaining its name and fatality on that account, extended to England under the milder form of a popular catarrh. Our influenza of 1762, to which dysentery succeeded, was remarkable, in comparison with those that followed it, for its severity and 'inflammatory type.' It was probably only part of the same malady which in the canton of Berne—taking shape from the soil on which it rested—raged with such virulence as to destroy life in four-and-twenty hours. Similarly the influenza that appeared in England in the spring of 1710 was the tail of the destructive pneumonic epidemic in Provence and Languedoc following the unequalled severity of the great winter 1708–9. We are not without evidence, indeed, that the last notable influenza outbreak of our own country—that of 1837—nearly resembled the great continental epidemics in its anatomical features. 'In most of the cases examined,' wrote Dr. Graves, quoting his own experience, 'both lungs were affected, easily breaking down under the fingers, and the portion so torn *resembled gangrenous lung except that there was an absence of fœtor*.'

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[1] The increased mortality of those times arose not from the influenza proper, but from the other diseases which prevailed along with it. Dysentery, diarrhœa, measles and (in the later epidemics) cholera, were its frequent attendants, and mark out those years as generally unhealthy. Yet all epidemics of influenza were not alike in this respect; succeeding influenzas varied considerably, as did different localities during the same epidemic. Thus the tendency to pneumonia, and the consequent mortality, was much greater in 1762 than in 1775. Yet in the latter year, in one city—Chester—inflammation of the lung was 'a frequent and dangerous termination.' In the epidemic of 1782 a single village was conspicuous for the inflammatory character of its symptoms. 'It now and then,' says a report of the College of Physicians, 'degenerated into peripneumony, yet these affections manifested themselves as particular modifications of the epidemic; they either preceded or accompanied some of its characteristic symptoms.' In regard to the influence of locality a remark of Hamilton's may be quoted to the effect that 'influenza partook more of pneumonia in dry and open situations.' With this may be compared an observation of Manetti concerning a pneumonic epidemic in Florence, that it was confined to the most airy parts of the city. See Dr. Theophilus Thompson's 'Epidemic Catarrh,' p. 109 *et seq.*

In modern times, as we shall presently show, pneumonia in epidemic form has ceased to be common, or perhaps it would be more true to say that the modern epidemics are less severe than the old, and on that account less notorious. Moreover, the affection is now more rigidly defined. In the period to which we have chiefly referred, pneumonia scarcely owned any anatomical definition whatever. Passive engorgement was always taken for inflammation, collapsed or compressed lung was never recognised as such, and pulmonary changes belonging to the mere act of death were interpreted as the signs of active disease.

Breaking through this darkness at the close of the last century came the discoveries of Avenbrugger and Bichat. The one proposed, though he did not perfect, a method by means of which the physical state of the chest organs should be gauged ;[1] the other, many years later, demonstrated the minute-structure of the lung. With the ground thus prepared and the work already begun, Laennec, pursuing the path which Avenbrugger had pointed out though he failed to follow it, proposed to take note of the actual condition of the chest organs by the audible report which they gave of themselves. The achievements of auscultation do not concern us here except in their bearing upon pneumonia. In the enthusiasm begotten of a new discovery, Laennec sought to reach and define by its means the essential signs of a disease whose boundaries and relations were as yet quite unfixed. By this new test, as he believed, an affection which had hitherto eluded description might be identified under a great variety of external forms. Pneumonia, although it could not always be seen, might by the aid of the new science always be heard. What really distinguished it was not the fever, nor the dyspnœa, nor the spitting, but *le râle crépitant.* This sound was always present, and, rightly apprehended, it was pathognomonic. The opinion thus expressed was eagerly caught up, and found an echo beyond the Channel. The crepitant râle, it was repeated, 'gives the earliest and surest intimation that such a disease has begun as tends to disorganisation and the inevitable loss of life, unless quickly arrested by its counteracting remedy.'[1] Thus a particular sound (whose precise mechanism is even now doubtful) was trusted to give reliable information which was to be credited though all the other signs might conspire to contradict it. The instructed ear could correct the inference of the other senses.

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[1] Avenbrugger's work ('Inventum novum ex percussione thoracis ut signo abstrusos interni pectoris morbos detegendi,' published in 1763) met with less attention than it deserved. According to Laennec's account, the author would seem to have been somewhat tardy and unenthusiastic, keep-

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ing silence for seven years 'inter labores et tædia.' Corvisart revived his views thirty years later. Bichat's 'Anatomy' was published in 1801. See Laennec, 'Traité de l'Auscultation,' vol. i. p. 27.

[1] The quotation is from Dr. Latham's 'Clinical Lectures,' and was repeated with approval in Sir T. Watson's Lectures forty years ago; a striking illustration of the revolution of opinion that time has wrought, not only in reference to the matter in hand, but still more in regard to the natural tendency of pneumonia and our views as to its rational treatment.

It is in accordance with all our experience of new methods to find them pressed at first for a larger service than they can render. It needed the observation of more than one generation to perceive that the function of auscultation had here been exaggerated. The crepitant râle, as it then appeared, might be heard in various circumstances, was common to many conditions of lung, and could never of itself announce with certainty the presence of a particular disease. Trusting entirely to physical signs, Laennec undervalued other sources of information, and those more obvious symptoms of pneumonia which had been already recognised. Yet it is to him that we owe the earliest picture of its successive stages. Improved means of research have since made evident much that was then obscure, and enabled later observers to define the pathology of lung inflammation more fully than he did ; but the clinical account of pneumonia, although it has since been amplified and refined upon, remains substantially as Laennec wrote it.

The labours of those who followed in Laennec's footsteps, of Andral, Grisolle, Chomel, Stokes, Williams, Addison, and others, belong to our own day, and form part of that abundant material, out of which a consistent history of pneumonia has to be constructed. The records of the past, as we have just now glanced at them, are perplexed and contradictory, yet there runs through the ancient accounts, no less than the modern ones, two leading characters. There is the widespread and often desolating epidemic associated with some unhealthy conditions of living, such as famine, bad lodging, and overcrowding, and bringing in its train other affections like dysentery and scurvy ; and there is the more strictly local disease called by this same name, prevalent more or less according to weather and bodily exposure, which is less fatal than the first, and without its accompanying diseases. In ancient times, the long series of epidemics of the sixteenth century, of which we have spoken, represents the first, while certain isolated examples, such, for instance, as the pneumonia affecting the garrison of Philippsburg, the second. In our own day, the epidemic pneumonia described by Dr. Bryson in the overcrowded ship 'St. Jean d'Acre,' with its diarrhœa and dysentery, or Dr. Dahl's outbreak of a similar kind in the overcrowded prison of Christiania, together with many other examples of which we shall presently speak, variously described as 'pythogenic,' 'sewer-gas pneumonia,' and the like, represent the first; while the Icelandic epidemic in the rough winter of 1863, or that occurring in the draughty barracks of New Brunswick, exemplify the second.[1] And not only in symptoms, associations, and mortality is this distinction to be noted, it is seen as well in the separate methods of treatment, and especially in contrasting the employment and the results of blood-letting and other 'antiphlogistic' measures in the two conditions respectively.

[1] See Chapter XIII, Epidemics.

CHAPTER II

THE FORMS OF PNEUMONIA

Definitions by various authors—Clinical and anatomical forms comprised under each—Illustrations—Rough classification—Essential characters of acute lobar pneumonia.

A MODERN author[1] has sought to bring disease within the limits of terse, dogmatic description, heading his subjects with a short definition after the manner of the exact sciences. The definition of pneumonia is as follows : 'A disease expressed by severe febrile symptoms, which come on suddenly, attaining in a few hours a great intensity, and which undergo a no less sudden abatement or improvement between the fifth and tenth day.' We read subsequently of its well-marked stages, of their pathological significance, and of the morbid sounds belonging to each. The affection is described throughout with singular precision ; it enters upon various phases in a prescribed order, and each step in its course has its appropriate physical signs, the significance of which can be nicely appreciated. It would thus appear that, in a given case, the physician would not only recognise the stage the disease had reached, but be able further to give some account of its antecedent phenomena, and to forecast with confidence something of its subsequent history.

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[1] Professor Aitken, 'Science and Practice of Medicine,' art. Pneumonia.

But we are next told that, although this is pneumonia, pneumonia is not always of this form ; that it is wont to attack the lung insidiously and in the course of other diseases ; that, so occurring, its features may be modified in various ways—so far modified, indeed, sometimes as to escape recognition altogether. It is then called (and, in the case supposed, no one can quarrel with the name) 'latent pneumonia.' One author, angry with a disease which so escapes detection, speaks of it when it assumes this shape as 'a low, sneaking inflammation.'[1] Similarly, the writer whose definition we have quoted alludes later on to obscure and latent forms of attack which have no resemblance to his first description ; and Sir Thomas Watson appends to his graphic description of the disease the caution : 'All that I have hitherto been saying relates to acute pneumonia, as occurring in a previously healthy person ; but pneumonia having that character and so occurring is a much less common disorder than most persons appear to suppose, or than I formerly thought it to be.' 'Inflammation of the pulmonary substance,' he adds, 'is apt to supervene insidiously upon various disorders which are of every-day occurrence—upon bronchitis, upon phthisis, upon disease of the heart, and upon fevers, especially the exanthematous fevers.'

In the more dogmatic statements of Dr. Walshe we find the varieties of pneumonia, which are indicated but not described, arranged in a table.[2] From this it appears that in its 'secondary or intercurrent origin,' pneumonia depends on a list of eleven acute diseases—in which rheumatic fever occupies the first place, and acute diseases of the brain the last—and on a list of chronic diseases, in which pulmonary tuberculisation and cancer occupy together the first place and Bright's disease the last. We read further: 'Instead of running the ordinary course, with marked subjective symptoms, pneumonia may be completely latent. Pneumonia occurs in this form solely under circumstances of general physical debility.' The author concludes by reminding us that, in treatment, 'we must remember that the inflammatory character of the local malady is modified more or less seriously by the general state of the system.' 'It is exceedingly probable,' he adds, 'that various differences exist in the intimate constitution of many of the intercurrent pneumonias, though at present no absolute proof of the fact can be given.' Other authorities might be quoted who thus vaguely allude to declensions from the typical form of the disease. Thus Dr. Stokes speaks of 'typhoid pneumonia,' which he explains to 'include a variety of cases, seen more frequently in hospital than in private practice, in which, whether from the low state of the constitution, the complication with other local diseases, or the pulmonary affection being secondary to a general morbid condition, we find a pneumonia often more or less latent, and accompanied by extreme prostration.'[1] Dr. Todd,[2] besides the simple disease, used to speak of pneumonia complicating gout and rheumatism, as well as of strumous, typhoid, and traumatic pneumonia; and Dr. Bristowe speaks to-day of the 'many pathological conditions which favour the occurrence of the disease and make it so common in the course of a great variety of disorders.'[3]

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[1] See Jones and Sieveking's 'Pathological Anatomy,' p. 428.

[2] Walshe, 'On Diseases of the Lungs,' 4th ed. art. Pneumonia, p. 375 *et seq.*

All this seems but a perplexed account of the matter. First we are told at length of a disease which is characterised by certain well-defined symptoms. It is next intimated that these symptoms undergo modifications under various circumstances, that, in fact, the modified disease is far more common than the simple one. It soon appears, moreover, that by 'modifications' no less is meant than that the disease assumes an entirely new shape. The statement amounts to this : in certain cases, in a certain definite way, the lung becomes consolidated (inflamed, as some believe), and this change is accompanied by such and such symptoms ; but much more often the lung is wont to become consolidated, whether inflamed or not, in a different way and under different circumstances ; and although all these forms of lung-consolidation alike are called pneumonia, the description we give applies only to the least common.

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[1] Stokes 'On Diseases of the Chest,' p. 338.
[2] Todd 'On Acute Diseases,' p. 368.
[3] 'Theory and Practice of Medicine,' p. 403. 5th ed.

A sense of the unsatisfactory nature of such classifications is apparent in the very language of the authors, and in the changes and rearrangements to be found in successive editions of the same book. Dr. Walshe in his later editions finds it necessary to describe pneumonia no longer, as in 1844, as 'acute inflammation of the pulmonary tissue,' but less directly as 'the representative of a variety of pulmonary inflammation processes widely differing in significance, in nature, and in issue.' The acute pneumonia of the earlier edition becomes the 'acute sthenic exudative pneumonia' of the later ; a writer who does not squander words needing three epithets to identify the true disease and place it beyond equivocation.

If to these accounts there be added the opinion of the late Dr. Hughes Bennett, that 'there are cases with all the symptoms of pneumonia yet with no inflammation of the lungs, and others with pneumonia yet with none of its symptoms,'[1] the confusion of the subject will perhaps be sufficiently exhibited.

And, turning from books to clinical records, the obscurity does not diminish. Here, for example, are two subjects whose lungs are described as exhibiting the appearances due to pneumonia. The one died by gradual sinking, with symptoms resembling continued fever, and but little embarrassment of respiration; the other was seized suddenly with urgent dyspnœa and acute pain, and died in a few days, suffocated. Yet the disease, so far as the name indicates it, is the same in both—only 'latent' in the one, and 'active' in the other. Instances are numerous where the contrast is as striking. How far the pathologist of the present day, ignorant of the histories of these patients, might succeed in classifying them in a manner which would correspond with their several clinical features remains to be considered. The actual fact is that, in our records, every variety of disease would appear to be nearly associated with this common name—pneumonia. It attacks the subjects of chronic diseases of many kinds; it is a frequent attendant upon typhus and enteric fever; it occurs sometimes quite suddenly and unexpectedly as a 'complication' in acute rheumatism; while not unusually patients who have been sinking little by little, with slow and painful lingering, die at last of pneumonia 'in its latent form.' Only rarely does it fall with fatal force upon the healthy and robust; and then its course is so rapid, and its characteristics so marked and uniform, as to excite wonder that so definite a disease can admit of so many modifications and varieties.

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[1] 'Practitioner,' vol. ii. p. 263.

Some years ago, when the office of Medical Registrar at St. George's Hospital offered favourable opportunity for such an enquiry, an analysis was undertaken of all the fatal cases named pneumonia recorded in the hospital books during a period of twenty years. Proceeding on the ground that the clinical features of each case had claim to consideration no less than the anatomical, the problem was to ascertain, first, whether these examples of disease—unselected and, except for being entered under a common name, quite dissimilar—might be arranged into natural groups ; and next, supposing them to be so arranged, to ascertain, further, whether the clinical grouping corresponded with the pathological, so that, for instance, a case distinguished during life by certain declensions from the type of the acute pneumonia of the books might be known after death by corresponding declensions in anatomical respects. In other words, the question to be tried was this :—Is any natural clinical classification of these cases possible; and, if so, how far does such classification serve to bring together cases which are anatomically similar?

The result of this labour [1] led to the conclusion that all the fatal instances of so-called pneumonia occurring in this series of years fell naturally, in view of their clinical histories, into four classes. The *first* and largest class would comprise patients who died of tedious and exhausting diseases of whatever kind, such as the constant drain of an abscess, or the gradual extension of large areas of ulceration, as from bedsores ; or, generally, where lingering was unusually prolonged, and prostration extreme. A *second* class would consist of the subjects of a specific fever, or of some definite affection of a secreting organ, and conspicuously renal disease and the poison of typhus. In some of these cases the lung-affection gave rise to clinical symptoms, resembling idiopathic pneumonia, with which, indeed, it would claim, sometimes, a near alliance. In a *third* class hepatisation would seem due mainly to mechanical causes, such, for example, as would arise from defective power of the heart ; from obstacles opposed to the circulation owing to some valvular imperfection ; from the altered constitution of the blood itself ; or from any combination of these states. *Fourthly*, hepatisation was met with in that form which the prevailing nomenclature describes as 'acute sthenic exudative pneumonia.' It was then invariably associated with pleurisy and sometimes with pericarditis ; then, and then only, it ran a definite course, other organs besides the lung being sometimes involved.

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[1] It was impossible out of the material to get more than a naked-eye description of the affected lung, but to avoid the inclusion of irrelevant cases care was taken not only that the reporter of each case described it as one of pneumonia, but also that the lung should in every instance exhibit hepatisation. To avoid unnecessary complication, cases connected with tuberculosis and phthisis, or with secondary pyæmic deposits were excluded from the list.

Thus, of the four classes the last was the only one which could lay claim to distinctive features, or be classed and described among diseases as having a history of its own. Hepatisation, in the other classes, is an accident ; and, while some affections, no doubt, are more exposed to it than others in an order that can be stated, its modes of access and of conduct are so various as to admit of no general clinical description. And, moreover, although it be true that hepatisation is a feature common to all the classes, yet these are distinguishable, the one from the other, anatomically no less than clinically. No doubt there remain many examples of a complex kind to which it is difficult to assign an exact place ; yet this is no more than we should expect in accordance with our whole experience of disease which habitually evades precise and rigid classification.

The wide field that is in fact occupied by these categories, descriptive of a single disease, is best exhibited by actual examples. Take such an instance[1] as the following :—A middle-aged woman, always greatly distressed by vomiting during her pregnancies, finds that symptom occur in the fourth month of such a time, and with so much severity and persistence, that hardly any nourishment whatever is retained. After two months of this incessant sickness, she slowly sinks exhausted and starved to death. Whatever conjecture is formed as to the material cause of this result, certainly pneumonia is not thought of, since there was neither cough, nor pain, nor dyspnœa, nor other symptoms that could be referred to the lungs. Yet it appears that in this woman, who thus died inch by inch, enduring the want of food for more than two months, 'the lower parts of both lungs were red, solid, and airless, from the early stage of pneumonia.' This woman's disease, therefore, from the name given it, would appear to be the same with that which in six days sufficed to destroy a robust man in the prime of life. This latter, on the day of his admission to the hospital, was seized suddenly with severe rigors, acute pain in the right chest, fever, &c. The combination of symptoms led to a diagnosis of inflammation of the right lung. Upon this supposition—following the practice of the time—he is bled twice within a few hours, sixteen ounces of blood being taken each time. He is then ordered half a grain of tartarised antimony every three hours; and the report of the third morning states that he 'has been unable to sleep, owing to vomiting caused by the antimony; he is also much purged.' So the dose is reduced to a quarter of a grain. As to nourishment, there was as little as possible of it, and that little of the most unstimulating kind. He is next blistered; and though the blister, it is said, 'rose well, the man sank and died, violent delirium preceding death by a little, and giving place to insensibility. When this patient's body came to be examined, it was found that these energetic means had failed to subdue the inflammation, such as it was. The lowest lobe of the right lung was in various stages of red and grey hepatisation, and there was some recent pleurisy. The upper lobes of the right lung and the whole of the left lung are described as 'remarkably healthy;' nor could any disease be discovered elsewhere in the body.

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[1] In the first edition of this work the cases here alluded to were included in an Appendix. In the present work they will be found, with others like them, either in the Illustrative Cases, Chapter IX, or incorporated with the text among the varieties and associations of pneumonia referred to in Part II.

Take another case different from either of these. A cabman of twenty-two, who has had rheumatic fever four years back and suffered from palpitation ever since, catches a cold and hereupon begins to spit blood. Soon his legs begin to swell, the urine becomes scanty, and he suffers from breathlessness. After three weeks of this he comes to hospital. His ten days' residence there is marked by increasing dropsy, orthopnœa, slow, irregular action of the heart, and at last delirium and death. He, too, died of pneumonia. 'The whole of the lowest lobe of the right lung was in a state of red hepatisation. At its lowest part among the inflamed tissue was a small spot of pulmonary apoplexy, while the mitral orifice was narrowed and perfectly rigid.'

One more, a cachectic old woman, who for nine months has suffered from pyrosis and vomiting, but without cough, dies by gradual sinking after six days' residence, vomiting being the only symptom recorded. Pneumonia again. 'The lower lobe of the left lung is quite solid, red and grey hepatisation being intermixed, and a layer of recent lymph spread over the pleura.' The post-mortem appearance, therefore, is very similar to that of the man with acute symptoms and active delirium who was so industriously bled.

We have here so many illustrations of the several ways in which hepatisation is set up; examples not of the same disease exhibiting itself in different ways, but rather of different diseases having in common a single pathological feature. In the first case, that of the middle-aged woman, the hepatisation is the so-called 'peripneumonie des agonisans' of Laennec, the hypostatic pneumonia of Piorry; it is not the cause of death, but the mode of it, and but one of a number of signs indicative of a gradual failure of vitality. It is misleading, therefore, to call it by a name that suggests characters which, in this particular case, are conspicuous by their absence, while they are seen in perfection in the marked symptoms and speedy death of the subject of the second case. The third patient, the cabman, is an illustration of the third class, where hepatisation is greatly due to mechanical causes. Constriction of the mitral orifice tends to keep up a constant pulmonary hyperæmia. Upon the man's taking cold, the stress of his trouble falls, as usual, upon the faulty organ, and the ultimate hepatisation of the right lung, with actual effusion of blood in one spot, which is but a further stage of the hyperæmia observed in the left, is the consequence of a direct obstacle to the pulmonary circulation. Finally, in the instance of the old woman, hepatisation, which is only one event, hardly perceived in the general failure, is again the result of obstruction. Yet it is not, as in the preceding case, due to a strictly mechanical cause, but to defective elimination, owing to advanced disease of the kidney giving rise to uræmia, whereby the already enfeebled circulation is still further embarrassed.

How far these cases, and the groups of which they are the representatives, are, from a strictly histological point of view, to be regarded as fulfilling the definition of inflammation, may be discussed presently. All that we maintain now is that hyperæmia and hepatisation, either or both, are sometimes the consequences of pre-existing disease, and sometimes they are the expression of the disease itself. Taken alone, there is no strict clinical significance attachable to these conditions simply as such. In other phrase, there is a disease, pneumonia, of which the most prominent local manifestation is hyperæmia and hepatisation; and there is, besides, the anatomical condition of hyperæmia or of hepatisation, apt to supervene in the course of many diseases, and notably connected with the many causes which tend, mechanically or otherwise, to embarrass the pulmonary circulation.

This view of the matter, which has to be further justified in the sequel, suggests such a nomenclature as shall prevent a casual, dead resemblance being mistaken for an inherent and living likeness. Pneumonia, with all its surroundings, in its sharpness of attack, its pyrexia, its anatomical changes, definite duration, and associated pathology, must stand by itself. Post-mortem conditions, which resemble it in part at one or other of its stages, are not, on the assumption of a real kindred, to be allowed to take its name. The present nomenclature stands almost self-condemned. 'Acute sthenic exudative pneumonia' is quite too long a term for a disease which is at once simple and distinctive; while the various appellations of disparagement applied to its supposed associates, as 'false,' and 'low,' and 'bastard,' throw a doubt upon what is elsewhere asserted, and clearly indicate that the relationship is not legitimate.

In the following pages we propose to describe, under the name pneumonia, that form of lung inflammation which, whether fatal or not, is characterised by sudden onset, well-defined symptoms, distinct anatomical stages, and limited duration. Assigning to it the simple title, we shall have next to consider what degree of declension from this typical pattern as regards origin, course, anatomical features, and morbid associations, may be fitly included within the boundary that separates the disease indicated from the many conditions which, though essentially distinct, resemble pneumonia here or there. That done, and having considered the provoking causes and the sequelæ of the disease, we shall have the material for discussing its pathology; the statistics of mortality under various circumstances; and the indications thence derived for our guidance in those matters of supreme importance which concern prognosis and treatment.

CHAPTER III

CLINICAL HISTORY

Aspect—Onset—Cough—'Stitch'—Sputa—Urinary changes—Temperature—Pulse—Tongue—Abdominal and nervous symptoms.

THE aspect of pneumonia, its precise look, manner, and posture, less express than that of some other diseases, may not always be recognisable in the features. Yet a mere glance at the patient is often enough to discover it.[1] A flushed, perhaps slightly dusky face, with a wearied yet anxious expression; a dorsal position with indications of pain and restraint in breathing, which, out of mere feebleness, the patient does not seek to lessen by change of posture; prostration nearly resembling that of enteric or typhus fever, yet with a less blunted perception, more hurried respiration, and the look of pain from pleuritic stitch. With more obvious distress than is commonly seen in enteric fever, it is distress that chiefly betrays itself in the face. The restlessness and contrivances of posture in order to still pain which we see in simple pleurisy are usually absent.

If a patient presenting such an appearance is approached more closely, other symptoms will become evident. A hot skin, dry and pungent like that of typhus or scarlatina; a pulse not unlike, it may be, in softness and volume to the pulse of continued fever, yet differing from it in that it is not accelerated in a like degree with the acceleration of the respirations. In fever, from whatever cause, there is quickened breathing, but in pneumonia this will be more marked, not merely from the hindrance to free inspiration caused by the pain of pleurisy, but also because the lungs, though as yet, it may be, without the formed products of the disease, are already disturbed in function in anticipation of the material changes which are impending.

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[1] In this general description the subject contemplated is a young adult. The comparative frequency of the disease at various ages, its mortality in the old and in the young respectively, and certain peculiarities it exhibits in childhood and infancy, will be considered presently.

With such an aspect and posture,[1] a temperature register between 103° and 104° or higher, and this altered ratio between pulse and respiration, it would hardly need more to determine the character of the disease. The patient would tell of a recent and sudden attack of shivering and sharp pain in the side, which, he will probably refer, rightly or wrongly, to some external cause. He is a groom, who first felt a chill when riding against the east wind, or a sailor, who caught cold while working in the hold of a draughty ship, or a labouring man, who on his way to church was suddenly seized with shivering on facing a blast of cold air.[2]

We say that the symptoms just detailed, with the knowledge that the attack was sudden and recent, and marked by distinct rigor, would suffice of themselves to signalise pneumonia without having recourse to special means of investigation. It may even be said that the affection is sometimes indicated by these characters at a period when its physical signs are as yet absent. Instances of such a mode of invasion have been recorded by various observers from the time when pneumonia was first formularised, and when, as has been said, its diagnosis was regarded almost exclusively from the physical point of view.[1]

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[1] A patient with pneumonia sitting up in bed, or lying with the shoulders much raised, will probably have *more* than pneumonia—sometimes effusion into the pericardium, or a dilated heart, sometimes a large quantity of fluid rapidly effused into the pleural cavity.

[2] These are all actual narratives of patients, illustrating the absolute suddenness of attack. A man admitted to hospital with well-marked pneumonia described at the time the exact place on Westminster Bridge where his disease had met him ; leaving the Surrey side of the water well, he entered the Middlesex side with it.

As a rule, however, it is not only by the signs now mentioned that the advent of pneumonia declares itself. There is the cough and the material that the cough expels. Upon the first it is hardly necessary to dwell; cough is precarious and changeful, liable to be influenced by many accidental circumstances, and of itself hardly an aid in determining the diagnosis. Commonly it commences within the first twelve hours of the illness, is short and infrequent, and chiefly a source of annoyance from the deepened inspiration to which it gives rise, augmenting the pleural pain. Paroxysmal cough, such as is common in bronchitis, is here quite exceptional, as is also the substernal pain which is among the earliest troubles of that disease. But, while cough more or less is rarely absent,[2] the gravity of pneumonia cannot be measured by its character or severity. A short, ineffectual cough, early disappearing altogether (as seen sometimes in the aged) is not of good but rather of bad augury, while violent and paroxysmal cough, except with children, is almost always an indication of the bronchial mucous membrane being extensively engaged.

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[1] We shall have to recur to this point presently. We would here refer to the kind of testimony alluded to in the text. Andral relates the case of a man of thirty-three, of full plethoric habit, who experienced the general symptoms of the disease, head-ache, general debility, numbness of the intellectual faculties, with a flushed face, injected eyes, and a frequent full pulse, at least six days before the stethoscope could detect the first appearance of the lung affection. 'All the organs,' says Andral, 'seemed to be simultaneously the seat of a strong excitement, without there being in any part real inflammation. No organ was really inflamed, but all seemed to be on the verge of it, as if they were all disposed thereto by too rich and too stimulating a blood.' 'In some cases,' says Grisolle, 'I have noticed for four days intense febrility without being able to discover on the part of any organ, and especially on the part of the lung, any lesion capable of explaining it.' In some instances, as this author thinks, though very rarely, the local inflammation would thus appear to be no more than the consequence of the antecedent fever. Andral, 'Clinique Médicale,' case 12; Grisolle, 'De la Pneumonie,' p. 187.

[2] Grisolle noted cough at the commencement of 80 out of 90 cases; the exceptions occurring chiefly in aged people. *Loc. cit.* p. 209 *et seq.*

A more valuable sign is furnished by the *expectoration.* It is, indeed, in some respects the nicest and most instructive of them all, for it reveals no less than the material part of the disease in a shape so distinctive as to be rarely misleading. Sometimes pneumonia is without spitting ; children under the age of five years seldom know how to spit, and grown people, especially the aged, may be too much enfeebled by the disease to make the effort. In such subjects the recognition of pneumonia is sensibly obscured.

The chief characters by which pneumonic expectoration is distinguished are—colour, consistency, and coalescence. The sputa, which are not abundant, are from their viscidity spat out with some difficulty, and appear in the porringer as one trembling, translucent, jelly-like mass, quite odourless, of an almost homogeneous consistence, and a colour like the rust of iron, whether the shade be deeper or lighter. Not uncommonly the little mass which accumulates in the course of six or eight hours is so viscid that the vessel containing it can be inverted without disturbance of the contents ; but sometimes there mixes with the proper expectoration of the disease a sufficient amount of watery bronchial secretion and of mere spittle to loose the proper pneumonic sputum from its hold or to prevent it from cohering into a single mass. A similar result may be due to the accident of the porringer containing water.

A uniform colour does not always pervade the mass. Often in the course of a single day, or between day and night, the precise degree of blood colouring to which this sputum owes its distinctive character will be seen to vary. Thus at one time it will be a dark reddish brown, finely streaked here and there with blood, or even with little clots of blood speckling it; at another like amber, or again of a faint rose tint, or hardly perceptibly coloured. But what is especially to be noticed, independently of these accidents, is this—that a material whose consistence is denser than that of the proper bronchial secretion, and which is neither mere mucus nor mere pus, is intimately mixed up with the colouring matter of the blood, while the blood itself sparingly escapes.

Blood-streaked sputum, such as is seen in obstructive heart disease, is quite different from this; the viscid, vitreous, quite uncoloured sputum met with in some forms of bronchitis is also different. The pneumonic sputum is not streaked, but tinted or stained, and, although the characteristic colouring of blood may be absent for a time, although it may become greenish, or may commence as a bright saffron yellow, there is always, or almost always, some indication that blood is exuding.

At what period should this sputum appear, how long should it last, and what changes does it undergo with the successive stages of the disease? There is no want of statistics upon these points, but it is only the first that can be answered with any degree of precision. In the majority of cases the pneumonic sputum is distinctive as to colour and consistence with the early symptoms of the disease. Yet it is not the very earliest, nor is its evidence always convincing. All expectoration may be delayed till the third or fourth day, or, appearing sooner, it may be at the first watery and bronchial, and only by degrees become coloured and viscid.[1] Sometimes expectoration is suppressed, or, as it were, diverted, owing, as is supposed, to the occurrence of profuse sweating or watery diarrhœa; sometimes (as in a case mentioned by Chomel) it is not suffered to appear, owing to mere ignorance of the art of spitting.

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[1] In hospital practice the details of the earliest symptoms have to be obtained from the report of the patients. In M. Grisolle's experience of 131 cases, 45, or more than a third, exhibited characteristic sputa on the first day; 76 (considerably over a half) did so on the second day; in six cases expectoration was delayed till the sixth day; and in one it was as late as the eleventh. The same author observes that the anomaly of uncoloured sputum in pneumonia relates to certain epidemic constitutions,

Absence of expectoration is sometimes ascribed to the particular seat of the affection. Pneumonia affecting the upper lobe has been said to show this among other peculiarities, the reality of which we shall have to discuss presently. It is enough to observe now that the cases quoted in support of this statement have exhibited as well extreme prostration, a condition which of itself sufficiently explains the anomaly. The theories once raised to account for the absence of spitting, where the apex is alone involved, upon physical grounds, have long since been disposed of.

We may not say, then, that a characteristic sputum is always the accompaniment of pneumonia, nor even, without reserve, that when this kind of spitting is present pneumonia necessarily exists along with it. There is a viscid kind of nasal mucus which, in some instances of inflammation of the pituitary membrane, becomes uniformly tinted with blood. Passive congestion of the lungs, of mechanical origin, will sometimes put on a similar appearance. But the resemblance is here less close, and resides rather in colour than consistence. Such sputum is seldom tenacious and jelly-like.

Again, some accidental admixture of colouring matter other than that of the blood may alter the character of the expectoration: ineffectual cough or a sensibility so blunted that cough is not provoked may prevent its appearance. But while recognising such exceptions, it is the rarest event to find the

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and was particularly observable in the influenza year 1837. (Grisolle, p. 217 *et seq.*) Chomel found the sputum white and frothy in three cases only out of 125 (Chomel, 'Pneumonie,' 183).

sputa retaining throughout a purely bronchial character. Rusty sputum in one or other of its degrees is, in the adult, among the main distinguishing features of pneumonia.

Microscopic examination reveals, as might be supposed, numerous blood discs, and, together with these, oil globules, granular cells, altered epithelium, pavement and columnar, and, sometimes, moulded fibrinous casts of the ultimate bronchioles. So far, therefore, there is little that is distinctive, except, as we shall show presently, that during the height of the disease there may be detected large numbers of diplococci in the sputum, when this is treated by special methods of staining (Chap. XVI, Bacteriology).

Similar catarrhal products are to be found in the sputa of acute bronchitis. Even the moulded casts, which might seem appropriate evidence of exudation (and have been so regarded by Remak and others), are found sometimes in bronchial inflammation, insomuch that 'plastic' bronchitis takes its name from their presence. But while pneumonic sputum exhibits itself mainly by its consistence and colouring, it is recognisable chemically from containing an excess of the fixed salts, and especially of chlorides; from the absence of alkaline phosphates; and an altered relation between its soda and potash.[1]

Coincident with these phenomena, and supplementing them, are certain obvious *changes in the constitution of the urine.* They may be divided into those which are common to the pyrexial state, as diminution in the total volume of urine, increased density, increase of urea and uric acid; and those which are peculiar, or, more truly, are most strikingly manifested in this particular affection, namely, diminution or total absence of the chlorides and the presence of albumen.[1] Later on, the crisis of the affection is marked by the sudden, abundant, often excessive, return of these chlorides, in correspondence with the equally sudden disappearance of pyrexia.

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[1] In Bamberger's observations, as quoted by Dr. Wilson Fox, pneumonic sputa are said to be characterised as follows:—

1. They contain no alkaline phosphates, while catarrhal sputa contain 10 to 14 per cent. of alkaline earths.

2. In catarrh the soda is to the potash as 31 to 26, while in pneumonia it is as 15 to 41.

3. Sulphuric acid in catarrh is equal to 3 per cent., in pneumonia to 8 per cent. At the period of 'resolution' the chemical character of pneumonic sputa approaches the catarrhal type. (Reynolds' System, vol. iii. 'Pneumonia,' 628.) Dr. Walshe has detected sugar in pneumonic sputa.

The urine of pneumonia resembles in fact that of typhus most, and its analysis would correspond closely with that made by Dr. Parkes in a case of that disease where, with diminution of water, the urea was increased one-fifth, the uric acid was in large amount, and the chlorides entirely absent.[2]

The elimination of urea from day to day is subject to variations irrespective of those in the body temperature. The *total urea* during the fever (according to Dr. West's observations)[3] is below the normal, and markedly reduced when compared with the quantity of nitrogen ingested. The destination and disposal of this lost nitrogen, its retention in the body or elimination 'in some other form than urea,' must remain for the present matter for speculation.

It would appear from the inquiries of Dr. Lionel Beale that the urinary chlorides, absent, as has been said, during the acute stage of the disease, are eliminated through the lungs by means of the sputa.[4] When expectoration is very slight the channel of elimination is presumably the bowels. In such circumstances the occurrence of spontaneous diarrhœa during convalescence is thus accounted for. Again, certain exceptional cases of pneumonia where, while the urinary solids are lessened, the chlorides fail to reappear, are exceptional further in the protraction of recovery. It is supposed by Dr. Parkes that these are instances where the tissue metamorphosis is delayed, or its products are retained in the system to the damage of the individual.

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[1] In rheumatic fever the changes are of like character, albuminuria excepted, both urea and uric acid being increased, but to a less extent, the chlorides diminished and sometimes absent, but 'the diminution is neither so great nor so constant as in pneumonia.' The same may be said generally in reference to acute pleurisy, but it must be added that the urinary changes are here often remarkably slight, and the rule elsewhere preserved of pyrexia running parallel with excessive formation of urea 'altogether breaks down.'

[2] See Parkes 'On the Urine,' p. 258.

[3] 'Observations upon the Elimination of Urea,' by Dr. Samuel West, communicated to the Medico-Chirurgical Society, November 10, 1874.

[4] This observation of the deportment of the chlorides was first made by

The frequent occurrence of *albumen in the urine*, and not of it alone, but occasionally of blood and fibrinous casts of the uriniferous tubes, is a significant fact in the pathology of pneumonia. Apart from diseases directly affecting the kidneys there is no acute affection save diphtheria and typhus, so often associated with albuminuria. We have here one among many indications, to be noticed more fully in the sequel, that the disease implicates other organs than the lungs, and affects the system to a degree not always commensurate with the local affection. Solon, who first called attention to the symptom, was certainly mistaken in connecting it with the period of convalescence. It is rather at the height of the disease that albumen makes its appearance, although from a multitude of

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Redtenbacher, who asserted, what cannot now be maintained, that the disappearance of the chlorides marked the precise period at which hepatisation took place. Neither is this so, nor do the chlorides reappear with resolution, but some days later. Dr. Lionel Beale ('Med.-Chir. Trans.' vol. xxxv.) has added to these observations those mentioned in the text with reference to the destination of the chlorides.

Dr. Beale points out that the method usually followed, and which was that employed by Redtenbacher, of estimating the chlorides by adding a few drops of nitrate of silver solution to acidulated urine, gives a rough estimate of the chlorides both fixed and volatile ; a precipitate may thus be due to an excess of chloride of ammonium when chloride of sodium is deficient or even absent. The usual clinical method, therefore, is so far fallacious, and the few cases of pneumonia where it has been assumed that the presence of a precipitate so obtained indicated the presence of chloride of sodium, do not necessarily bear that interpretation. *Vide* 'Med.-Chir. Trans.' p. 327, vol. xxxv.

observations it would appear impossible to associate the condition always with any particular stage of engorgement or consolidation on the part of the lung.

There is, indeed, considerable disparity, as Dr. Parkes points out, in the accounts of various observers of the frequency of albuminuria in pneumonia, statistics varying from nearly 45 per cent. to nothing. Metzger failed to find it once in 48 cases; Dr. Wilson Fox found it 10 times in 32; Ziemssen only twice in 24; but Griesinger in more than half, namely, in 63 out of 121. What most strikes the observer, next to their remarkable disparity, is the smallness of these figures. Dr. Dickinson is led to believe from his own experience that the larger estimates are the more correct, so far as relates to true lobar pneumonia; and inserts a table, supplied by Dr. Isambard Owen when Medical Registrar at St. George's Hospital, whence it appears that of 26 cases of pneumonia, albumen was found in all but four. Even in these latter, it was not held certain that more frequent examination might not have discovered it.[1] 'The albumen appeared at all times between the second day and the tenth, and no fixed relation held between its date of appearance and that of resolution.'

The same author goes on to state that the pneumonic albuminuria resembles that of tubal nephritis, save that the urinary solids appear to be increased, with the exception of the chlorides. He believes, however, that œdema is 'practically unknown' as a result of this form of albuminuria, nor is he 'aware that persistent renal disease has ever been traced to this beginning.' Moreover, the opinion is expressed that the urine becomes albuminous at that early period of the disease when the essential urinary excreta are in large excess and the urinary water deficient. The urea in particular may be increased to more than twice its normal amount, and this at an early stage of the pneumonic fever antecedent to the absorption and discharge of its inflammation products. 'It is not improbable,' writes Dr. Dickinson, 'that the kidneys owe their disturbance to the demand thus made upon them, the attendant irritation enhanced possibly by the want of water.' [1]

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[1] 'Renal and Urinary Affections,' p. 1255.

In a casual examination of this particular symptom the error may be committed of attributing every instance of albuminous urine to the then existing pneumonia, forgetting that the subjects of albuminuria due to renal disease are especially prone to lung inflammation. With patients seen for the first time it needs care, repeated microscopic examination, and an attentive survey of the previous history, in order to estimate the exact place and significance of this symptom.

The precise prognostic significance of albuminuria in pneumonia is open to question. Dr. Parkes (on the basis, however, of a very small number of cases) states the mortality of patients with albuminous urine at 50 per cent., with non-albuminous urine at 14 per cent. He quotes Finger, who, out of 15 albuminous patients (the time of first appearance of albumen not being stated) lost 6, or 46 per cent.[2] Among 98 patients of our own albumen was found in the urine in 27, but in by far the majority it was present in very small amount, and often quite transient in its appearance. In three cases, however, it was considerable in quantity, and lasted for some time, but eventually disappeared. It must be observed that in many fatal cases, dying within 24 hours of admission, the urine was not obtained; but it is noteworthy that of the 27 cases in which albuminuria existed, five were fatal, whilst among the 71 in which its absence is noted, there were but two deaths (or a comparative mortality of 18·5 per cent. against 2·8 per cent.).

A full review of the whole matter in reference to albuminuria in pneumonia inclines us to the opinion that the severer cases rarely fail to exhibit albumen in the urine at some period or other, and that thus this particular symptom gets associated with unfavourable indications; but we hesitate to affirm that the discovery of albuminuria in a given case taken by itself adds to the gravity of prognosis.

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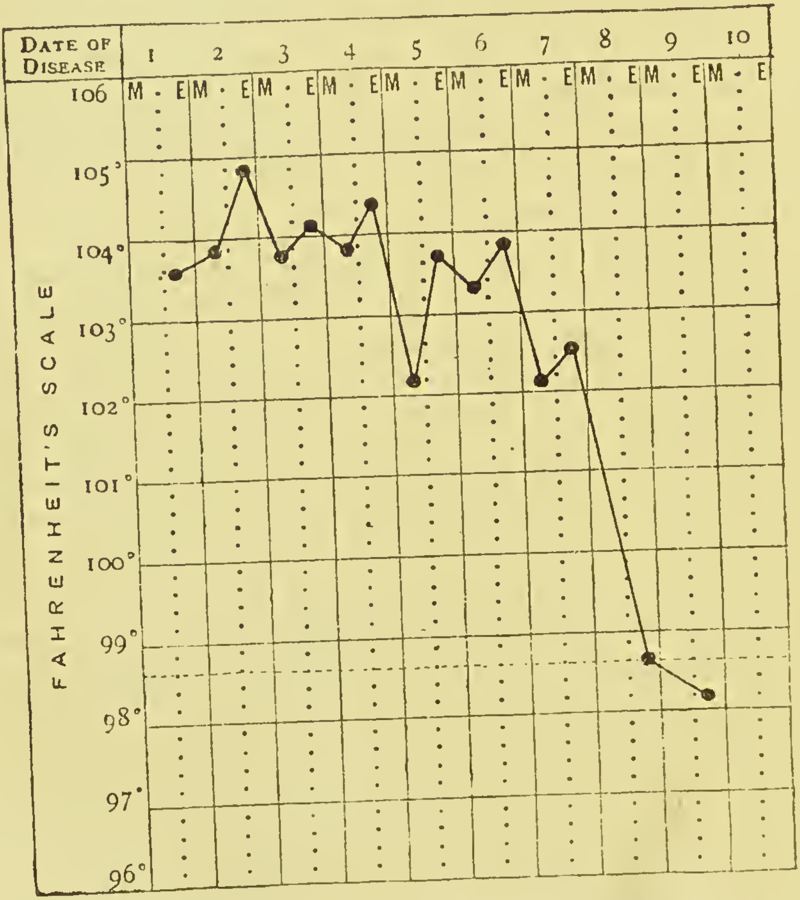
[1] *Loc. cit.* p. 1259.

[2] Parkes, *loc. cit.* p. 277.

CHART I.



Coming next to *pyrexia*; the pneumonic temperature is high at the first, that of the evening usually somewhat above that of the morning; fairly sustained, yet with irregular variations; and at the end of its term—most commonly on the seventh day—undergoes an abrupt fall. The earliest temperature may be the highest, but at any time of day or night the body heat may remit for a few hours or minutes with no corresponding change in the patient's general condition. It is common, indeed, on the fourth or fifth day, to observe a remission so decided as to be mistaken for the favourable crisis, whose advent it is never safe to predicate on the showing of temperature alone. These points are well indicated in Wunderlich's typical chart-tracing given above (Chart 1). It may be added that, a day or two after crisis, temperature will often rise irregularly for a time without interrupting the course of recovery.

Hyperpyrexia is more rare with pneumonia than with acute rheumatism, yet the former fever may reach 107° and the patient recover. The temperature may register 105° or higher at the outset, and shortly decline without further indications of danger, while a very moderate pyrexia sometimes attends that spreading form of pneumonia which is so often fatal.[1] It must be added that the thermometer gives no certain information, either as to the extent of lung involved, or the probable duration of pyrexia.

That it is easy to place too much reliance on the evidence of temperature alone is clearly shown by comparison of temperature charts as nearly as possible similar, yet referring to patients in all other respects unlike. Below (Chart 2) are two examples of adult pneumonia side by side. The one, represented by the dotted line, was a woman of twenty-one; the other, represented by the continued line, was a man of twenty-seven. The three days shown in the chart correspond with the most acute stage of illness in both patients. One recovered, the other died; one was constantly delirious, the other had no delirium at all; yet upon mere inspection, it is impossible to say which is which.

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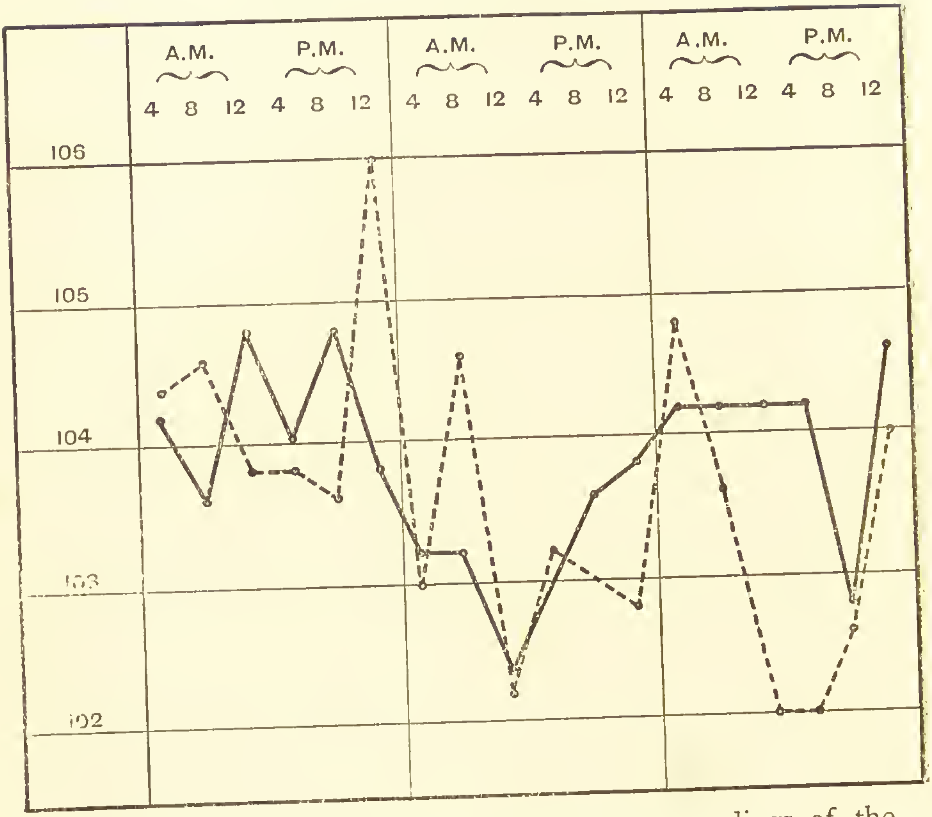
[1] See Case XII, Chapter IX.

CHART 2.

LOBAR PNEUMONIA—4-HOUR TEMPERATURE.

Robert S., æt. 27 (continued line).
Elizabeth L., æt. 21 (dotted line).



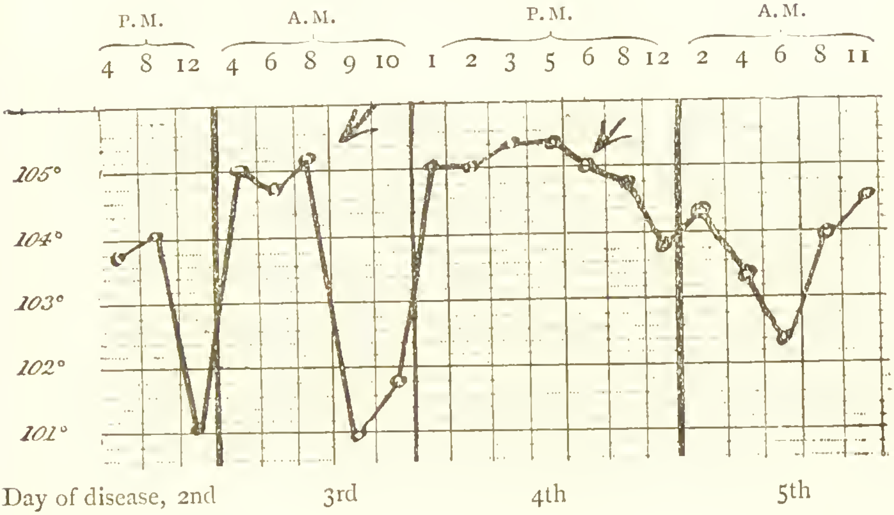
The dotted line shows the temperature readings of the second, third, and fourth days, crisis happening on the seventh with rapid recovery. The continued line represents the temperature on the eighth, ninth, and tenth days, of a pneumonia fatal on the twelfth day.

In children especially, a critical defervescence may precede ascent of temperature and a fatal end. Here, for example, is a copy of the pyrexia of an infant under a year old, a rare instance of fatal lobar pneumonia at that early age. Twelve observations were taken for one day, viz. from 4 a.m. to the following midnight, as represented in the chart; the child dying on the fifth day of the disease.

CHART 3.

LOBAR PNEUMONIA—2-HOUR TEMPERATURE.

Edward A., under 1 year.



High temperature on the fourth day, was both preceded and followed by what might be taken for critical fall.

With children, as with adults, a fatal pneumonia may exhibit but a moderate register throughout. A tracing in shape very similar to the above is now before us: that of a young child whose temperature (taken every 2 hours) at its highest was only 2 points above 102°. Similarly Dr. Peacock[1] reports a case of pneumonia fatal on the sixth day, and examined after death, where the highest temperature, 102·5, was reached on the fourth day, from which time till death it fell rapidly, just as it might do in a favourable crisis, reaching 97·6 very shortly before death. Other examples of like kind will occur in the course of this work.

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[1] Peacock's Report on cases of Pneumonia, 'St. Thomas's Hospital Reports,' vol. ii.

A high temperature at the beginning of pneumonia does not *ensure* a severe attack; a sudden elevation may be ephemeral and does not always *imply* an increase of the disease; a stationary, or even a falling temperature, *may* concur with a rising pulse and increased frequency of respiration; one patient may die with a temperature never exceeding or reaching that of another whose illness has given no occasion for anxiety.[1]

The temperature charts of typhus, quinsy, and facial erysipelas may nearly resemble that which is characteristic of pneumonia in degree, duration of pyrexia, and critical descent. From many examples we quote the following temperature tracing (Chart 4) of quinsy in a girl of seventeen. The observations were made with remarkable assiduity during sixty-six hours, forty observations being made in that time.[2] Both in shape and range it nearly agrees with the pneumonia chart already given.

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[1] Some anomalies in pneumonic temperature were reported many years ago by Dr. J. G. Bacon of Saratoga in the 'New York Medical Record,' as follows:

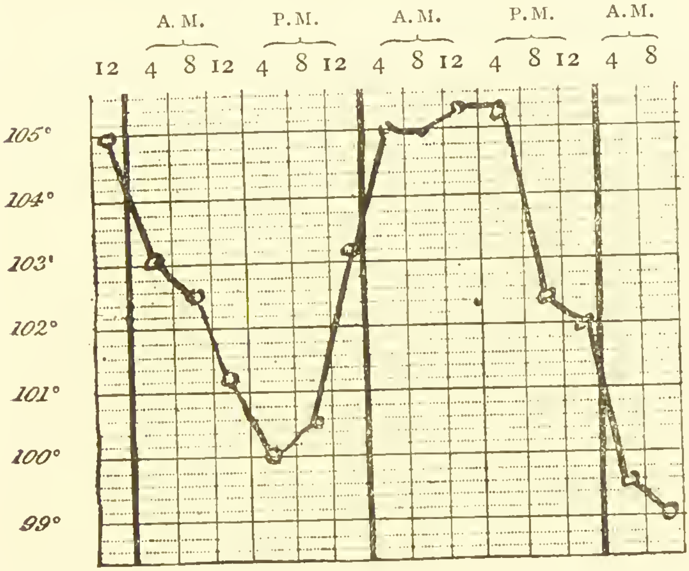
'A girl aged 16 years, strong and full-blooded, had pneumonia of the left lung. Temperature began rising up to fifth day, when it stood, as carefully noted by a self-registering thermometer, 107·5°. The sixth day it fell to 104° by evening. The condition of the patient otherwise showed no cause for alarm, as far as consultation could decide. She is now about, and free from cough, and gaining her strength fast. The second case was her brother Frank, aged 20 years. He was seen twelve hours after he began to complain. Pulse 165, wiry. Slightly delirious. Examination revealed pneumonia of right lung. Temperature, very carefully taken thirty-four hours after I called, was 110°. I could not believe my eyes until I had repeated the experiment several times with the same result. The expectoration was nearly pure blood for forty-eight or fifty hours: cough harassing. Now, here is a strange feature (to me) in this case: the temperature was 110° at 5 p.m., and the next morning, at 9.30 a.m., it had fallen to 99°. An intense diaphoresis occurred, which continued for twenty hours.'

[2] For the saving of space this case is here represented on an abbreviated four-hour chart. This serves well enough if it be kept in mind that the joining lines correspond very nearly with *the actual observed path of the temperature*; they are not, as with a four-hour chart, merely hypothetical, nor, as with a morning and evening chart, purely imaginary, and only certain of being wrong. Those who content themselves with a morning and evening reading of temperature and no more, might at least have the grace to omit lines of juncture to such isolated observations; these cannot fail to be misleading.

CHART 4.

QUINSY—HOURLY TEMPERATURE (ABSTRACT).

Susan G., æt. 17.



Pyrexia sometimes persists for some days after crisis.

In some instances, not otherwise exceptional, temperature will remit considerably each morning or evening for several days before the actual crisis (Chart 5).

The case which this chart illustrates had, however, other exceptional features, such as insidiousness of onset and absence of rusty expectoration. It was also of interest from the fact that nine months previously the patient had had a somewhat similar attack, with a like course of pyrexia. The case is given in fuller detail in Chapter IX,[1] Case VIII.

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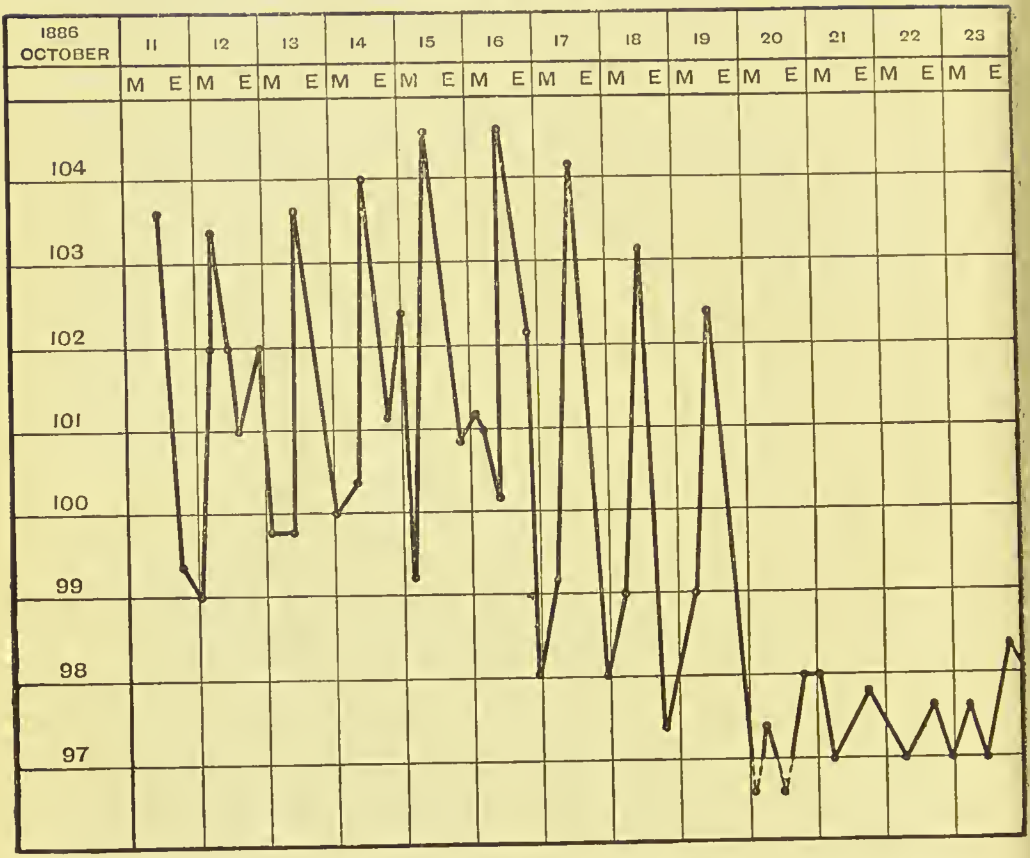
[1] For similar variations in the temperature curve in pneumonia, see a paper by Bertrand, entitled Des Anomalies du Type Fébrile dans la Pneumonie Fibrineuse, 'Revue de Médecine,' July 1889.

CHART 5.

Lobar Pneumonia—Remittent Pyrexia.

Emma H., æt. 28.



A descending temperature,[1] concurring with 'critical' sweating, may sometimes give fallacious appearance of recovery, as in the chart (6) below of a pneumonia fatal on the seventh day. (*See* Case XII, Chap. IX.)

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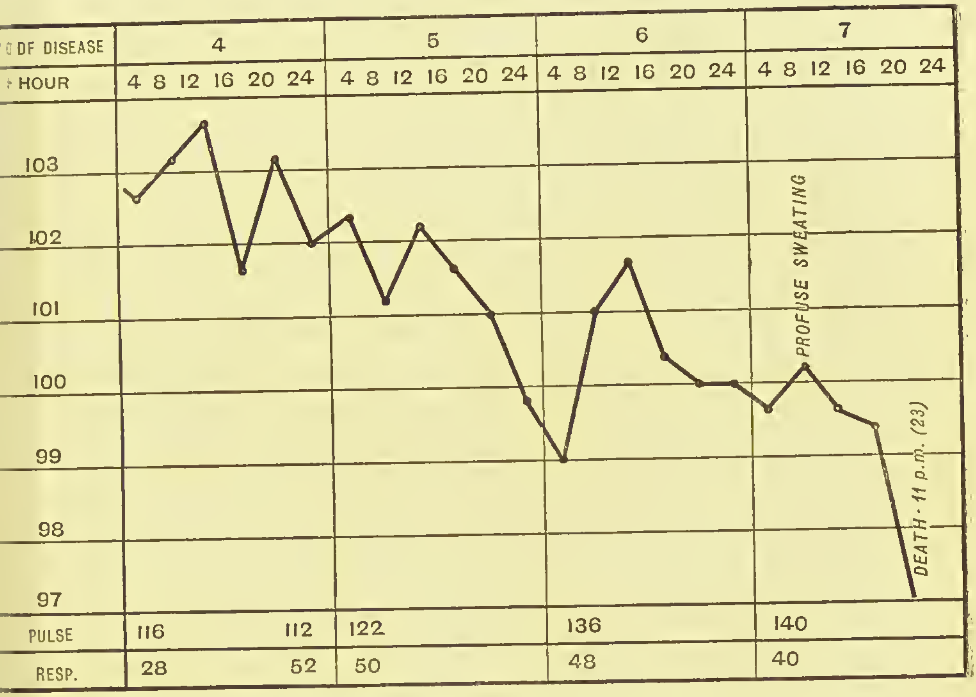
[1] In a large number of cases, as we shall presently show, temperature falls quite gradually throughout, insomuch that the tracing gives no indication of crisis, notwithstanding that the patient's amendment is sudden and at the usual time. Thus a robust man, aged 31, was admitted to the Westminster Hospital (March 9, 1890) on the first day of a lobar pneumonia of right lung. His *highest temperature was on the following* (*second*) *day*, 104·2°. On the third day, when the physical signs were still developing, the tracing was already in descent. By the sixth day it had fallen by gradual lysis to 99°, respirations having daily increased in frequency from the first, that is, from 40 to 80. On the seventh day, with temperature

CHART 6.[1]

Henry C., æt. 42.



Notwithstanding the foregoing cautions and exceptions (which will find further illustrations in other portions of this work) it would be untrue to say that the thermometer is of small use in this disease. Like other isolated signs it is not a certain guide, it may mislead the unwary and must never be trusted alone. One of its chief services, perhaps, in pneumonia is to distinguish that disease from other forms of lung consolidation which resemble it in physical signs.

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still 99°, breathing fell to 48, and thence to 32, and the following, or eighth, day marked the term of the illness.

[1] The twenty-four hours are here numbered progressively, a convenient plan obviating the possibility of confusion between days and nights.

Much greater reliance is to be placed on the *pulse*. A pulse that *keeps* over 120 in the adult male, whatever its character, must always cause anxiety. Wunderlich would call all cases 'severe' where the temperature records 104°, the pulse more than 120, and the respirations are over 40 during the height of the disease. But until further defined in terms of death rate, the word 'severe' is quite arbitrary. According to Griesinger, one third of those with a pulse over 120 die; an observation which, like other computations of the same sort, is of little value in view of the changeful character of pneumonia at different times, with different subjects, and in different epidemics. Whatever its rate, it is commonly soft (differing in that respect from the pulse of pleurisy), and often, yet not persistently, dicrotic, owing to the sharp propulsion of blood with diminished peripheral resistance. As stated by Walshe, in old age, pneumonia may be present with no acceleration of pulse whatever. In exceptional cases it may be but slightly quickened even in the young,[1] while on the other hand with children it may be so frequent as hardly to be counted. A pulse small, and of weak beat, but no great frequency, may concur with very grave symptoms. With soft, non-conducted mitral murmur and prominent jugular veins, such a pulse is indicative of over-distended right ventricle, a temporary condition tending to produce partial paralysis of this chamber. With its continuance, the left ventricle in its turn gets ill supplied, and the blood sent forward is deficient.[2] There results forcible action of the heart in marked contrast with small, weak pulse and concurring with lividity of face and distended jugulars, a condition which, as we shall see, venesection relieves for the while to a remarkable degree.

An intermitting pulse, according to Juergensen, is of worse augury than one of great frequency. In our experience, intermittence, apart from cardiac disease, is a very rare occurrence at the onset of the disease.

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[1] See Case IX, Chapter IX.

[2] See Broadbent, 'The Pulse,' p. 194, &c.

The *perverted ratio of pulse to respiration* has been much insisted on, particularly at that early stage when, owing to the absence or obscurity of physical signs, an important element of diagnosis is wanting. Taking the normal ratio of respiration to pulse as 2 : 9, a sudden transformation of that expression whereby the respirations approach and may even exceed the pulsations is among the early signals of pneumonia, and may anticipate any physical evidence of it by more than twelve hours; and although it be true that acute pleurisy, acute phthisis, and other chest affections, alter the proportion in a similar way, yet in pneumonia the perversion is the greatest, insomuch that early in the disease the respiration-pulse ratio may possibly exceed 1 : 2.

The value and the need of this test for recognising pneumonia will be considered presently. It may be pointed out now that it is chiefly to be relied on in those cases where the accompanying pleurisy is without much pain. Pleuritic 'stitch' gives rise to shallow inspirations, and these in young and nervous patients may become very frequent, making up in rate for what they want in individual efficacy; but the quickening of respiration, which in pneumonia produces a similar but more extreme perversion of the respiration-pulse ratio, is independent of pain and an intrinsic sign of the disease.

As regards *the digestive organs*, the indications furnished by the tongue, abdomen, and alvine discharge are not characteristic at the commencement. As in other febrile states there is anorexia, while in some cases obstinate refusal of food is among the chief difficulties of treatment.[1] Constipation, according to Juergensen, is met with in three-fourths of the cases; but with young children diarrhœa is not infrequent and vomiting very common. This latter symptom, indeed, appearing at an early stage together with convulsion (in these subjects the equivalent to rigor) may lead to a diagnosis of meningitis which is presently shown to be erroneous as well by its sudden disappearance as by the advent of distinctive physical signs within the lung.

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[1] See Case VI, Chapter IX.

The tongue, although in rare instances hardly altered from health, is usually white-coated. The thickness of this covering varies from the 'stippled' to the 'plastered,' while sometimes prominence of the papillæ will give the 'strawberry' tongue.[1]

It is in the later stages, and not at its commencement, that the evidence of the tongue becomes valuable; but in rare instances, and especially where the upper part of the lung is involved, a dry tongue may concur with marked prostration at an early period of the disease.

From the occasional prominence of gastric symptoms, vomiting, jaundice,[2] and hepatic tenderness, some authors have recognised a special form of pneumonia, but examples of the kind conform in other respects to the common type and do not, in fact, require a name of their own. Enlargement of the spleen is said by Juergensen to be an occasional accompaniment of pneumonia, an observation which, in common with most physicians, we have not been able to confirm.

A distended and tympanitic abdomen, at the commencement and before the appearance of physical signs, may not only be the source of considerable discomfort,[1] but also confuse the diagnosis between pneumonia and enteric fever. In children, especially when the apex is the part attacked and there is drowsiness, but no stitch, respiration not greatly quickened and no expectoration, the uncertainty may not be wholly dispelled until the crisis. Observation as to the character of temperature : the absolute suddenness of pyrexia ; and in most cases, but not in all, the perverted pulse respiration ratio, together with the absence of characteristic typhoid motions, may succeed in detecting pneumonia in this disguise (Chapter XII).

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[1] In Dr. Dickinson's classification ('The Tongue as an Indication in Disease,' pp. 33–55) the tongue was 'not abnormal' in 2 cases of commencing pneumonia out of a list of 24 representing a great variety of diseases; it was 'stippled' (*i.e.* with excess of fur not uniformly spread, but displayed only on the points of the papillæ) in 6, whether pneumonia or broncho-pneumonia, out of a similar list of 62; 'coated white' in 6 pneumonias out of 48 diseases of many kinds; 'strawberry' in 2 out of 7, its company being scarlatina 3, typhoid and perityphlitis 1 each; 'plastered' (the uniform thick-coated tongue of acute disease, which, losing moisture, becomes the 'encrusted' tongue) in 8 out of 32, the others being, typhoid, 6; other acute febrile conditions, 4; acute rheumatism and acute bronchitis, 3 apiece.

[2] See Chapter VI, p. 110.

Last in place, yet probably first in importance, of the early signs of pneumonia, we place its *nervous symptoms.* The *impression* that the disease makes at its first onset, the kind and the degree of nervous change that it brings, as indicated by aspect, gesture, and mental condition—these, most of all, measure the gravity of the peril that awaits the patient, not with absolute certainty, indeed, yet better than either pulse, respiration, or temperature taken singly. Apart from active mental disturbance and other nervous associations of the disease, of which we shall speak later, that kind of prostration which sometimes likens pneumonia in outward appearance so closely to enteric and typhus fever, belongs undoubtedly to its severer and more fatal forms.

Delirium is of many sorts, and depends to some extent on the idiosyncrasy of the patient ; but it is a symptom to be closely observed in pneumonia, and its varying significance in prognosis may be roughly classified. In patients addicted to alcoholic excess, this symptom, accompanied as it usually is by sleeplessness, is always serious, and the more so the more it approximates to the character of delirium tremens. Extreme restlessness without active delirium in these subjects is, perhaps, equally unfavourable. On the other hand, violent delirium at the outset of an attack will often quiet down so soon as the local signs are fully developed and be followed by pneumonia of no great severity. Such active delirium in young persons, where the seat of inflammation is the apex, is sufficiently common and express to deserve separate mention.[1] In rare instances, again, it is not at the first, but during crisis, that delirium occurs; the patient, under the spell of some delusion, shouting wildly and struggling with his attendants, at the same time that temperature is in rapid descent and the disease on the eve of departure. (See Crisis, Chapter IV, p. 60.)

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[1] In a child of seven with pneumonia, under the care of one of us, tympanites was so extreme as to cause serious embarrassment to the respiratory movements. It was but slightly relieved by hot fomentations and the insertion of a tube into the rectum ; drug remedies failing altogether.

Apart from active delirium, and associated with a latent form of pneumonia which has its origin chiefly in poverty and privation of food, is a settled mental derangement, during which the sufferer may wander about the streets, or attempt his life, or commit some act for which he is clearly not responsible.[2] Of this insidious form of the disease, ending commonly in rapid spoiling of the lung and death, altered mental state is, as we shall see, sometimes the sole symptom observed (Chapter X).

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[1] See Cases IV and V, Chapter IX, also Chapter X.

[2] A man of middle age admitted to the Westminster Hospital, had been discovered in the area of a house and arrested by the police. He could give no account of himself, and was obviously very ill. On admission to hospital he was found to have pneumonia with physical signs already fully developed. After a very severe illness he eventually recovered, retaining, as he averred, no recollection of his adventure or arrest, a statement which, apart from the fact of his disease, his character and antecedents, gave reason to credit.

CHAPTER IV

PHYSICAL SIGNS AND PROGRESS

Stages of inflammation as physically indicated—Minute crepitation—Bronchial and tubular respiration—Vocal resonance and vibration—Times of occurrence of hepatisation, early and late—Middle period between onset and crisis—Days and events of crisis—Physical signs of resolution—Convalescence—Modes of death.

THUS far we have designedly delayed the consideration of those features of pneumonia upon which it is too much the custom to concentrate attention, and which concern the physical condition of the lung itself. Yet with such a patient as has just been described it would be obvious, even to an unpractised observer, that the breathing apparatus was directly implicated. And, in fact, the lung, or some portion of it, is at this period, or is about to become, the seat of a morbid process which, by its successive changes, serves to indicate the stages and determine the duration of the disease.

This process, to speak generally, commences with hyperæmia, and is completed with the exudation of fibrin and escape of blood into the pulmonary air sacs. These two events, first an effusion of serum into the vesicular structure of the lung; next, the passage of a coagulable plasma which moulds itself in its new residence so as to consolidate the heretofore spongy texture of the organ, are distinctive of pneumonia. Yet they are not two, but rather stages of a single process. An exudation, at first watery, is ever becoming more and more coagulable as more and more of the material of the blood exudes through the vascular walls, while, in places, by little ruptures, the blood itself pours out. It is thus both an exudation and a hæmorrhage.[1] The rate of its progress, the variable duration of its several stages, and the effect produced thereby upon the lung-structure, constitute whatever modifications the disease legitimately admits of.

Now, the auscultatory signs, the sounds that meet the ear from day to day as the disease progresses, are in accordance with these events. At first, with the earliest effusion of watery serum into the vesicular structure of the lung, there arises, as the inspired air reaches the ultimate bronchules—at the end, that is, of each long-drawn inspiration—a *minute crackle*, not loud, but distinct, breaking in abruptly upon the soft-sighing of inspiration.

The verbal description of sounds is of doubtful service. No more can be done than by likening them to other sounds which are more familiar; even then the comparison does not seem equally apt to all hearers, and is of doubtful assistance to any. Nevertheless, the minute crepitation of pneumonia may be very closely imitated. A piece of quite dry tissue paper pressed up into a ball and squeezed in the hand with a varied amount of force, so regulated as to give a finer and finer crackling sound, will at last represent exactly *the character* of fine crepitation. Alternate pressure and relaxation by the hand, in time with the rate of respiration, will even convey something of the general effect that the ear receives when listening at the chest itself, although in *fineness* it hardly equals the minute bronchule crepitus.

Again, minute crepitation is imitated to perfection, as Dr.

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[1] The above summary of the morbid changes in pneumonia is intended merely to render the description that follows of the physical signs intelligible. The anatomy of the disease will be discussed in the next chapter.

C. J. B. Williams first pointed out, by the sound which is produced by rubbing one's own hair between the fingers close to the ear. The illustration, like the other, has the drawback that in mechanism it is unlike that of pulmonary crepitus. By a very simple contrivance, however, a sound can be obtained which, both in character and causation, may be made to resemble pretty closely this form of crepitation. A fine sponge, dipped into a weak solution of gum, is lightly compressed within the hand ; upon the gradual relaxation of the pressure, a crepitus ensues which will be more or less fine with the degree of dilution of the gum solution.

It is necessary to refine somewhat in speaking of this particular sound, because there is a coarser variety which is of different significance and probably different mechanism. We shall have to speak of this latter in connection with the resolution stage of pneumonia. It is elicited by means of forced inspiration, and indicates probably the reopening by the violence of the in-current of collapsed lobules. It is in accordance with this view of its origin to find this coarser or larger crepitus disappear for the while, so soon as by a full expansion of the lungs the lobules have been once expanded.

It is different with the crepitus we are now concerned with, and which depends, probably, upon the unsticking of inflamed lung during inspiration. Unlike the other, it not only abides quite unaltered by cough, but, in most cases, its time and place are unvarying. It will occupy, that is, not the whole inspiration, but only the latter end of it. A single inspiration may, indeed, sometimes be obtained without it, by purposely shallow breathing on the part of the patient ; but the needs of respiration are such that (except where very acute pleuritic pain is present) a suspension of the sound from this cause will never occur naturally.[1]

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[1] The mechanism of this crepitant râle is matter of dispute, and Dr. Walshe has discussed it at some length. If produced by air passing

Of the first stage of pneumonia this may be the earliest, and even for a brief space, the sole physical sign. Whether or not some perceptible lessening of the natural resonance arises along with it is a point not worth dwelling upon.[1] Theoretically one supposes that it would be so. It is more to the purpose to bear in mind that any marked degree of dulness at this period (while as yet the small crepitus is unaccompanied by blowing respiration) will be probably due either to the interposition of thickened pleura or to effusion into the pleural cavity.

While this minute crepitus is necessarily associated in the mind with pneumonia, since it occurs amongst its earliest physical signs, pneumonia may exist without it, and it may exist without pneumonia. The absolute probative value of this sign has, in fact, been much exaggerated. It proves no more than that the ultimate bronchules contain a superabundant amount of fluid of whatever sort or consistence. Hence, in so far as it is a sign-proper at all, it is a sign-proper not so much of pneumonia as of pulmonary œdema.

With the presence, however, of pyrexia, of 'stitch,' and of rust-coloured sputum, the meaning of minute crepitation is not doubtful. But it is not always present; may appear early or late, and in any case does not long exist alone. Often, indeed, so soon as the crepitation is heard, there is heard along with it an altered character of breathing—it becomes *blowing*, *sniffling*,

through fluid, why is it *inspiratory* only? If due to expansion (or unsticking) of the parietes of the vesicles previously (*i.e.* before inspiration) glued together, why is it sometimes to be heard with pulmonary œdema where the fluid is watery and not sticky? The latter, however, in spite of this objection, seems the most probable supposition. (See Walshe, *loc. cit.* art. 'Auscultation,' p. 110.)

[1] Skoda maintains that the percussion sound remains unaltered (Markham's translation, p. 289). Dr. Walshe believes, on the contrary, that it 'acquires to a slight extent the characters of Type I, the amount of tone diminishing, the pitch rising.' He adds, 'Any serious change in these respects renders it possible that an intermingling of actual exudation has already occurred.'—Walshe 'On the Lungs,' p. 350.

and soon tubular, or even markedly metallic—while, at the same time, the percussion note elicits absence of lung resonance, and the hand laid upon the place discovers exaggeration of the natural vocal thrill as the patient speaks. Such signs are the direct and sufficient evidence of solidification of lung by the filling up of its vesicular portion. The larger tubes only remain patent, while the increased density of the parenchyma surrounding them modifies (in many ways, owning many names) the sound of the entering and retiring air, so as to liken it to the blowing of wind through a pipe, the true tubular breathing of consolidated lung.

By this consolidation not only is the natural vibration of the lung with the act of speaking increased, but the voice of the speaker is conveyed to the listener's ear with a ringing clearness and intensity which will be more or less striking according to its natural tone. The phenomenon becomes at once apparent on comparing the solid lung with its fellow, and is most distinct with a bass voice. We are not to expect it in the high-pitched notes of women and children, while it is to be remembered that both the voice and its vibration—both *vocal resonance* and *vocal fremitus*—may be deadened or lost by the interposition of a quite thin layer of semi-fluid lymph. The voice in that case, consolidation notwithstanding, instead of striking the ear from its shrillness, may be distant, or quavering, or truly ægophonic. Such combination of physical signs is, perhaps, most characteristic of all, indicating, as it does, not only lung consolidation, but pleuritic exudation as well, which, as we shall presently see, usually accompanies it.

By keeping in mind the physical condition on which these signs depend, the observer is prepared for their occasional absence and various modifications. There are certain phenomena, however, in the acoustics of this stage, which, while they are sufficiently frequent to need mention, are still awaiting explanation. It will happen, for instance (and in cases not otherwise exceptional), that the solid lung, instead of yielding to percussion the usual dull, wooden note, gives a resonance[1] which suggests the neighbourhood of an empty cavity, deadness of sound, that is, along with tympanitic ring, or else that note which has been well called 'metallic.' The impression thus conveyed by percussion may be still further confirmed by auscultation. Cavernous respiration will often concur with this metallic ring, while the conclusions to which such signs point may be yet further strengthened by the existence of pectoriloquy.

Whatever may be the true explanation of these signs,[2] depending, as Dr. Gee believes, on the existence of islets of unsolidified lung in the midst of hepatisation, it is quite certain that they may be met with in lung that is simply consolidated. In the stage of pneumonia with which we are at present concerned, such abnormal sounds would not be really misleading. True pneumonia breaking up thus early, so as to produce cavity, is a very rare occurrence; it is also one which would be supplemented by a group of symptoms themselves not likely to be overlooked. There may be other anomalies,[3] not in the sounds themselves, but in the order of their occurrence. Not seldom, for instance, it will happen that minute crepitation is not heard at all at any period;[1] at the very outset the lung, or a portion of it, is found to be solid; the signs of hepatisation not being preceded, so far as is known, by those of engorgement.

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[1] Sub-tympanitic (Skodaic) resonance will be alluded to under 'Complications.'

[2] See Gee 'On Auscultation,' p. 254; see also Walshe 'On the Lungs,' p. 352, and under 'Percussion' and 'Auscultation,' upon the variations in character of bronchial and tubular breathing and their probable significance.

The terms bronchial and tubular are sometimes used indifferently, but the distinction is obvious and important. It is true, however, as we believe, that carnified lung with pleural fluid interposed will sometimes yield tubular breathing indistinguishable from that of hepatisation; true also that in the course of resolution the true tubular sound gradually alters to bronchial, and that in the course of this transition the respiration for a while partakes of both characters.

[3] A not infrequent source of error arises from the presence of pleural effusion along with pneumonic consolidation. The lower lobe of one lung solidifies, and at the same time fluid is poured into the pleural cavity. Now, the solid portion of lung being incapable of compression, the action of this accumulating fluid is to displace it, pushing it up against the superior and unaffected lobe, which thus becomes compressed and in time carnified. Although such a condition is quite recognisable, both before and after

A too strict divison of pneumonia into distinct parts, each having its own period and proper signs, equally with the assertion of a constant correspondence between pulse, respiration, and temperature, gives an appearance of conciseness at the cost of accuracy. The stages described as successive are often concurrent. Early in the affection the lung will sometimes exhibit to post-mortem examination every phase of change from hyperæmia to grey hepatisation.

In hospital practice, whence statistics are mainly derived, patients seldom come under observation at a very early period. Yet it is a frequent experience to find the breathing distinctly bronchial concurrently with the first occurrence of crepitation. It is rare, indeed, for this latter to be audible without some such

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death, yet it often doubly misleads. The displaced yet solid lung is, during life, taken by its sounds for a lung compressed merely, and the carnified upper lobe, *post mortem*, is taken for lung in an earlier stage of inflammation than the lower.

[1] In some rare instances a patient will present all the signs, physical and general, of pneumonia, save and except that over the portion of lung which, by percussion or other signs, appears to be consolidated, there is no breath sound audible, whether tubular or other. With the existence of the general symptoms of pneumonia such indications suggest the presence of fluid. This condition may be due to the obstruction of mucus, in which case cough will probably remove it. When permanent, it depends on the sealing up of a portion of lung by fibrinous coagulum, the so-called 'massive pneumonia' of the French as in the following case.

A man of forty-five was admitted into the Westminster Hospital with pneumonia. He had dulness up to scapular spine on the right side, but breath sounds were inaudible at the base, while higher up on that side they were indistinctly heard and vesicular. The patient was too ill for the voice signs to be employed. Exploration was made by the house physician in the expectation of finding fluid, but without success. The man lived less than two days. Post mortem: the greater part of the right lung was hepatised, red, turning to grey, pleura covered with soft recent lymph. The bronchi (even as far as the main right bronchus) were plugged with yellow, fibrinous coagulum.—*Post-Mortem and Case Book*, vol. v, p. 354.

change being detected in the breath sounds of the larger tubes, although a longer period may be needed before true tubular respiration is heard. Grisolle believed that in two-thirds of his patients crepitation was mixed with tubular breathing from the third day, while he has 'established the existence of bronchial breathing hardly twelve hours from the commencement of the pneumonia.'[1]

Instances of the early occurrence of tubular breathing will be referred to hereafter. They are met with especially in the intercurrent pneumonia of acute rheumatism, where it is common to find physical evidence of consolidated lung almost as soon as the former disease is recognised. On the whole, it may be said that the physical signs of consolidation suggest, though they do not prove, that the inflammation is more than twelve hours old; while it is to be remembered, on the other hand, that all physical signs whatever of pneumonia may be delayed until several days after its initial rigor.

The physical signs afforded by the chest organs in pneumonia do not refer to the lungs alone. The conduct of the heart and blood-vessels as the inflammation progresses needs examination also. Of the pulse we have already spoken in the previous chapter. As for the heart, it may be mentioned that, while lung consolidation never displaces that organ, it may serve to convey its pulsations to the hand of the observer, while, at the same time, conducting the cardiac sounds with extra distinctness. It is by the sounds of the right heart rather than the left that the degree of pulmonary resistance is best measured. The right ventricle's systole, shorter and perhaps louder than that of the left, is heard from the mid-sternal line to within an inch of the apex beat, but best over the lower costal cartilages, from the left edge of the sternum upwards and outwards for about an inch, while its second sound, as is familiarly known, is most audible over the pulmonary valves in the third left interspace close to the sternum. Given a healthy heart, not overpowered by resistance (as it may become under excessive strain), and the observation of intensified first and, still more, accentuated second sound in these situations will serve, *cæteris paribus*, to measure pulmonary obstruction, and this, as we think, not so much by the absolute character of these sounds at any one time as in the comparison of one day with the next in each particular case.[1]

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[1] Grisolle, *loc. cit.* p. 295. Dr. Wilson Fox says: 'Bronchial breathing may not appear until the second or even the fourth day, and this appears more commonly with pneumonia of the apex.' He mentions 12, 24, 48 hours as possible times. Walshe does not allude to this point, nor does Chomel.

It will be pointed out in its place that in the case of children consolidation signs may be scanty and dubious, bronchophonic cry being sometimes the main physical evidence of undoubted pneumonia.

It remains to consider what further symptoms mark the progress of a disease whose advent is thus plainly declared. At an early period in its career there comes an interval, not indeed of pause—for pyrexia persists, urea discharge is in excess or defect, the respirations and pulse are accelerated, and often there is albuminuria—but of obscurity, when the further course of the disease can only be faithfully ascertained by means of auscultation. For the changes now taking place within the lung, changes which are a part of the natural history of the disease, do not announce themselves visibly or by obvious signs. The passage from engorgement to consolidation, the spread of the disease in local area, even the implication of the fellow lung, are events which only physical examination can detect.

Now, too, comes the time of suspense, a sort of *middle period* between development and crisis, when considerations concerning age, habits, and previous health (to be dealt with in a future chapter) will assist prognosis no less than the actual condition of the patient at the moment. Yet, while these pulmonary changes are still in progress, the variations of body-temperature, the rate and ratio of pulse and respiration, and, above all, the face and manner of the patient, must be carefully scrutinised. From any of these quarters the first signal may come either that the stress of the storm has passed, or that it is about to overwhelm its victim. In the majority of cases, between the third and the eighth day from the initial rigor, a sudden lull comes. The temperature of the body drops by several degrees, and the pulse falls along with it, insomuch that sometimes in the course of forty-eight hours both may be below the standard of health. At the same time, and as suddenly, the febrile aspect is lost, and the habitual expression returns.

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[1] See Broadbent, 'The Pulse,' Chapter III. 'Heart Sounds in Relation to the Pulse.'

Up to the very eve of this event the temperature may have been in uneven ascent, or it may have maintained an equable or nearly equable rate for like periods of successive days; or again, occasional dips in the temperature tracing may have given for a few hours delusive hope that the fever had reached its term (Chap. III). Upon the occurrence of the real crisis the fall is apt to be considerable, six, eight, or even ten degrees in the course of a day and night. The pulse usually declines in frequency at the same time, but it does not fall so often as does the temperature below the normal rate. Sometimes its rate will vary from day to day with no corresponding variation of the thermometer, and sometimes becoming abnormally slow while the respiration is still frequent, owing to imperfect resolution on the part of the lung, the pulse respiration ratio is perverted to a remarkable degree. In some instances, as Walshe mentions, the fall of the respiration rate is considerable, and may even exceed that of the pulse, insomuch that the perverted pulse respiration ratio, upon which he so much insists, may wholly disappear. Concurring with these striking symptoms lithates may appear in the urine and the amount of urea diminish. It is to be observed, however, that these signs coincide rather with the anatomical stage of resolution than with the clinical phenomenon of crisis, and the two, as we shall see, are not always coincident.

Symptoms supposed to be critical (and no doubt rightly so in a large proportion of cases), such as diarrhœa and sweating, are sometimes, as the event shows, mere accidents, due to drugs or unsuitable diet; and sometimes they are the signals, not of recovery, but of impending death. Not less misleading may be a sudden fall of temperature, which, taken by itself, is of no particular significance and may be very short lived. Veritable crisis, the signs which betoken the turn of the disease and the safety of the patient, can never be safely predicted until the face takes part. Crisis may be expected on any day after the fourth; it may seem imminent from a falling temperature and a lowered pulse; but it is never assured until it is visible in the features. The sudden change of expression which indicates the very hour when the fever departs is perhaps the most striking of all the phenomena of pneumonia.[1]

The occurrence of crisis is not constant with pneumonia nor peculiar to it. In what proportion of cases it happens we will consider immediately. But in other affections and in specific fevers, notably in typhus, signs of amendment may be equally sudden, the fever quitting its hold as in a moment, whilst profuse sweating or diarrhœa or the discharge of highly lithatic urine supervening are regarded in the light of critical phenomena.

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[1] We might expect that a change in the character of the sputa would be among the earliest signs of the termination of pneumonia. It was so believed of old. Boerhaave, Cullen and Huxham only followed Hippocrates in supposing expectoration to be the natural mode of exit of the disease, and attempting to promote it by drugs. Later on, the belief in crisis by expectoration had the support of the elder Frank. Yet in truth the character of the sputa gives no more than a tardy indication of the local progress of the disorder. Crisis, as we shall presently see, often antedates the earliest physical signs of amendment on the part of the lung, and may be wholly completed while the sputa are still unchanged.

Critical fall of temperature, though indicative, as the event shows, that the disease has reached its term, is sometimes attended by symptoms of delirium or collapse in themselves dangerous.[1] It is as though the sudden drop of temperature (in rare cases, as has been mentioned, not less than ten degrees in the twenty-four hours) produced a shock of its own. Yet it must be added that such symptoms are exceptional, the rule being that the fall of temperature, even when considerable, coincides nearly with a general improvement.

We have said that this sudden disappearance of fever may be looked for between the third and eighth day from the initial rigor. It does not always fall within those days, but wide inquiry shows that the great majority of cases may be so included. Wilson Fox has collected statistics of several observers on this point. His tables show that in 374 instances as many as 324 had their crisis between the third and eighth day, the seventh and fifth being the favourite days.[2] Crisis on the ninth day is uncommon, and if longer delayed it is hardly to be looked for at all.

In our Report to the Collective Investigation Committee on Pneumonia (p. 47) 370 cases (or less than a third of the whole number) ended by crisis, and 298 of these fell between the third and eighth day, both inclusive. The seventh is the favourite day, comprising 85, the next largest number being on the fifth day, 59; and the days preceding and following the favourite, *i.e.* the sixth and eighth, being equal, 52 for both. After the eighth day there is a rapid decline. The ninth day shows a sudden fall, crisis occurring in but 29, hardly more than half the previous day, and not much more than the tenth, 21. For the eleventh day there is another great drop, namely, to 6, and the later figures are smaller still. This statement as to the entire number, 370, holds also in most respects when the cases are divided into three equal series, except that in one of the three the fifth is the favourite day.

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[1] In a case of our own, a man aged twenty-four, of temperate habits, whose crisis happened on the fifth day, temperature fell from 104° to 98° in forty hours. The disease had a favourable course throughout, and at its worst a pulse of 106, with respirations 50. Yet, while the temperature was in descent and when it had fallen to 101° this patient had delirium of such violence that extra help had to be summoned for his restraint. It was short lived, however, disappearing with the night, and he made quick recovery.

[2] *Loc. cit.* p. 650.

In the table below, side by side with the above figures, are the statistics of critical days according to Bleuler, Wunderlich, and Ziemssen, whence it will be seen that, with some remarkable discrepancies (especially in Ziemssen's sixth day), the seventh day is the favourite in every one.

DAYS OF CRISIS IN 698 CASES OF PNEUMONIA ACCORDING TO BLEULER, WUNDERLICH, ZIEMSSEN, AND THE COLLECTIVE INVESTIGATION REPORT.

Days	Bleuler	Wunderlich	Ziemssen	Collective Investigation Report	Totals
2nd	0	0	0	3	3
3rd	6	10	9	15	40
4th	13	11	3	35	62
5th	22	14	31	59	126
6th	26	14	5	52	97
7th	32	19	35	85	171
8th	24	4	4	52	84
9th	12	3	9	29	53
10th	6	0	0	21	27
11th	1	0	8	6	15
12th	3	0	0	3	6
13th	1	0	3	4	8
14th	0	0	0	1	1
Not stated	—	—	—	5	5
	146	75	107	370	698

Crisis, as we have seen, may be absent altogether. In 740 cases it was unnoticed in more than half. But no doubt sudden amendment, the equivalent of crisis, sometimes passes unobserved, because the body temperature fails to indicate it or indicates it uncertainly. Thus the thermometer may fall and rise irregularly for several days without ascertainable cause or apparent effect on the patient, or a lowered temperature resembling crisis on the chart may have none of the accompaniments of that event, and may even precede death.[1] Again, the concurrence of some other disease, such as acute rheumatism or bronchitis, may prolong the pyrexia period, as may also some of the occasional sequelæ of pneumonia, such as pleuritic effusion or empyema. The fact that irregular temperature changes often receive no explanation from the condition of the patient, and are no certain index of it, must be freely admitted. The service of the thermometer is not, therefore, to be denied ; it is isolated observation with neglect of the conjoined symptoms that misleads.

The doctrine of crisis falling by preference upon the uneven days was taught by Hippocrates, concurred in by Andral, and has since been revived by Traube. The discussion seems to belong rather to a past age of medicine, and we need not stop to enlarge upon it. Owing mainly to the frequency with which the seventh and next to it the fifth day mark the turn of pneumonic fever, there is no doubt that on the whole the uneven days are the more prominent in this respect. In the above tabular enumeration odd days number 413, even days 280. It is a matter of small importance. On the other hand there are two observations bearing upon the pathology of the disease which cannot fail to arrest attention. One is that the crisis of pneumonia often precedes, though it sometimes follows, the earliest physical sign of resolution ; the other is that the time and mode of crisis are not influenced by the extent or site of the local affection.[1]

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[1] See Case XII, Chapter IX.

An analogy has been supposed to exist between pneumonic crisis and the sudden defervescence which marks the completion of eruption in small-pox. Both events are supposed to illustrate the consequence of elimination from the system of a *materies morbi.* It is a pure hypothesis, and from what has been just said it may be doubted whether the comparison it invites is justified by clinical facts.

Hepatisation completed, the subsequent history will vary according to the nature of the exudation, as will be shown in the next chapter. But in any case the sealing up of the lung and abolition of breath sound from its vesicular portion marks the period when, for a while, we lose count of the precise physical changes that are in progress. These are not such as to modify at once the auscultatory signs. Between solid and semi-fluid, a hepatised liver-like lung, and one whose inflammatory contents are liquefying, there are differences of breath-note—transitions from tubular to bronchial—which the ear may mark. But between these extremes there are gradations of change which imply slight alteration in the density of the lung and own no separate expression in auscultation. The precise physical condition at this important juncture is mere matter of conjecture. The inflamed lung still announces itself to the ear as solid and airless, while the indurance of pyrexia beyond the period of expected crisis may arise either from accidental complications in no way injurious to the progress of resolution, or else be the herald of grey hepatisation and inevitable death.

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[1] In 72 out of 192 cases cited by Grisolle, a notable diminution in the febrile phenomena preceded by one or several days the stethoscopic changes; in 92 it was simultaneous with these, in 26 the stethoscopic signs of amelioration were the first. Thus crisis, so far as was seen, was non-simultaneous with the material lung changes in 98 cases—more than half. Some critical phenomenon or other coincided with resolution in 34 out of 130 cases. See Grisolle, pp. 300 and 311–315.

What then, it may be asked, is the exact degree of change in the consistence of the exudation which may be trusted as announcing that resolution has begun? Such a question, it is obvious, cannot be answered to demonstration. Yet it seems certain that the most constant and often the earliest intimation of commencing resolution consists in altered quality—lessened intensity, as we say—of the tubular breathing. Successive modifications of this tubularity may be traced sometimes through all gradations, from a metallic blowing as through an inflexible tube, to a respiration which is harsh merely, and wanting in the soft character of healthy vesicular breathing. Sometimes these transitions will be more abrupt, and sometimes their gradual character may pass unobserved; for it must be allowed that the phenomena of resolution, coinciding as they do with the safety of the patient, are followed with a less lively interest than those which attach to the active stages of the disease.

Along with this lessened tubularity, but more variably than it, occurring so capriciously, indeed, as to be of little practical value, is the coarse bubbling described by systematic writers as *redux crepitation.* This sound is chiefly of importance as having been sometimes mistaken for the minute crepitation of the commencing disease, and an indication of relapse.

Redux crepitation, however, is larger, coarser, more separate and more scanty, so to speak, than is crepitant rhonchus. Forced inspiration will sometimes alter both its character and its place. It is of very variable duration, may apply both to inspiration and expiration, and only in exceptional instances offers any near likeness to true crepitus. Dr. Walshe alludes to the vague manner in which the phrase redux crepitation is used, and has long been led to the conclusion that under that phrase two very different phenomena are confounded—a fine bubbling rhonchus and a true returning primary crepitation. The former is by far the more common, has all the characters of a humid rhonchus, and is, he has little doubt, produced in the minute bronchial tubes; the latter is probably generated in the same seat and manner as the primary crepitus.[1]

Though the physical signs of resolution are apt to be heard in the order stated, the tubular breathing becoming less tubular before either rhonchus or redux crepitus is heard, it would not be true to say that the process of recovery fulfils in an inverse order the process of the advancing disease. In the oncoming stages the most perfect tubular breathing may be announced quite suddenly; in resolution its departure is never really sudden. Again, the crepitation of resolution is quite uncertain in time, duration, and place of occurrence. Weeks after the patient has recovered it may be heard from time to time in deep inspiration, or, as with minute crepitation, it may never be heard at all from first to last.

Along with the other signs, and like them variable as to time, is a lessening in the brassy ring of the voice and a percussion note no longer absolutely dull. It is not uncommon, however, for dulness of some degree to remain for an indefinite time when the breathing is restored to its normal character, a condition due to a thickened pleura.

Corresponding pretty closely with the phenomena of resolution is the return of the urine to its natural constitution, the reappearance—sometimes in excess—of the chlorides, urea discharge[2] in normal quantity, and absence of albumen. The continuance of albuminuria is, however, variable and uncertain.

The convalescence of pneumonia is brief and secure, and recovery usually complete. Of its occasional complications and delays we shall speak presently. It is enough to say here that lingering convalescence is rare, and that in the majority of cases the patient is well in the third week.

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[1] See Walshe, 'Diseases of the Lungs,' p. 355.

[2] According to Dr. S. West's observations, already quoted, the total urea in convalescence is at first reduced and subsequently rises to that of health.

But there is another side to the picture. Pneumonia may destroy life in several ways. Either the inflammation products so embarrass the lungs as to interfere to a fatal degree with respiration, or the inflammatory process assumes a destructive character so as to spoil the organ it invades ; or, thirdly, the demands which the disease makes on the strength are excessive, and the patient dies exhausted. Of the tendency of the affection in any of these directions physical indications may tell but little. That pneumonia is grave in proportion to its area, and most fatal to those who are already enfeebled, though it may seem like a truism, is not even true. Of course, the danger of death by apnœa is heightened by the wide extent of the pulmonary inflammation ; but such cases are not necessarily severe. In that interval of suspense, when auscultation ceases for a while to be of much service, the general condition of the patient needs as nice a scrutiny as in enteric or typhus fever. Whatever the physical signs, urgent dyspnœa ; suppressed cough ; difficult, scanty expectoration, which clings to the lips and teeth, and is viscid, bloody, and, perhaps, prune-juice-coloured ; muttering of irrelevant matters ; a flushed and purpling face, and above all, progressive dulness of aspect and perception, these are evil symptoms often concurring, and it is needless to add always full of peril.

In such circumstances as we have said, our knowledge of the actual physical state of the lung—of the precise anatomical stage the disease has reached—is far from accurate. As a matter of fact it is usual to find it, after death, grey rather than red, and not seldom so disorganised as to cause wonder at the absence just before of distinctive auscultatory signs.[1]

[1] There is a form of pneumonia, nearly allied to acute phthisis, where the affected lung will so break down at a number of separate points or

Yet often near the end—it can hardly be said in anticipation of other signs whose meaning is not doubtful—there may be added to the physical indications of consolidation those of general pulmonary œdema. Crepitant rhonchus heard at one or both bases will signify the access of general congestion, a common preliminary of total failure.

In this sketch of the main features of pneumonia we design no more than to place before the reader the cardinal points of the disease. Certain details remain to be filled in, certain statements, perhaps, to be defended. But the general character of pneumonia is not matter for doubt or controversy. It is both a local inflammation and a general disease. As an inflammation it is remarkable for its distinct stages, for the character and conduct of its inflammation products, for the preservation of structure amid changes which in other organs are the prelude to permanent damage. As an affection of the general organism it is no less remarkable for the definite duration of a pyrexia which is not measured by the extent of the local inflammation or strictly synchronous with it. If the pyrexia is symptomatic, why is it not a truer index of the inflammation that provokes it? If, on the other hand, the lung affection is no more than the local expression of a specific fever, whence does this fever arise and how does mere exposure suffice to produce it in a healthy subject?

centres that auscultation is able to discover in the more advanced of these the existence of actual excavations, and the physical condition is thus accurately known before death.

CHAPTER V

MORBID ANATOMY

The anatomical stages of lobar inflammation : (1) Engorgement ; (2) Exudation ; (3) Liquefaction —Rare terminations—Abscess—Gangrene—Caseation—Induration—The anatomical characters of lobular pneumonia.

The knowledge to be gained concerning any disease from the appearances presented by the organs and tissues after death cannot but be partial and imperfect. Morbid anatomy and its younger sister histology do, it is true, reveal the structural changes wrought in the parts examined by perverted processes of nutrition, but neither scalpel nor microscope can demonstrate the actual manner in which such lesions have been brought about. That is purely a matter of inference and conjecture, founded in part on the comparison of the characters presented by the altered tissues at different stages of the malady in different subjects, in part on pure hypothesis and physiological reasoning. The inferences derived from the study of dead tissues may but vaguely suggest the nature of the morbid process, the evidences of whose operation are manifested in the symptoms observed at the bedside. Morbid anatomy, in fact, may render probable a certain sequence of events, which have resulted in the lesions displayed, but it can do no more than this. It is precisely the same with the study of normal structure and function. The circulation of the blood is suggested by the disposition of the blood vessels, the arrangement of the valves in the heart and veins, and the muscularity of the heart, but it is not proved thereby. It is not safe to predicate function from structure in the normal body, and it is even more hazardous to infer from structural lesions alone the possible history of the morbid vital processes, that have left their record behind them, since pathological physiology is still fragmentary and contains many unsettled problems.

The stages into which it is customary to divide the anatomical changes in the lung in pneumonia are four in number, viz. (1) congestion, (2) red hepatisation, (3) grey hepatisation, (4) resolution. These terms are sanctioned by usage, but the conditions might perhaps be more correctly described as follows :

1st stage : engorgement.

2nd stage : exudation, including (*a*) red hepatisation and (*b*) grey hepatisation.

3rd stage : liquefaction, often termed 'purulent infiltration.'

The process may occasionally be varied in its final periods by the formation of an abscess, or the development of gangrene, or else by a gradual transition into chronic fibroid induration.

Stage of Engorgement.—Instances of more or less localised congestion of the lungs, limited, that is, to a single lobe or to a portion of a lobe, such as may be inferred to denote the earliest stage of the pneumonic process, are rarely met with in the post-mortem room. At any rate, it is impossible to affirm of any such case that pneumonia was in progress. One explanation for their rarity lies obviously in the fact that, even in the most rapidly fatal cases, the lung, at the time of death, has advanced beyond this stage, and whatever may be said for or against the occurrence of a stage of the disease anterior to that of pulmonary engorgement, no one would expect that the reality of such a condition could be established anatomically.

In the highly improbable event of death at that early period it would remain for ever uncertain whether or not pneumonia had actually existed. Anatomically, therefore, the affection first becomes recognisable in hyperæmia of the lung. Yet even here, as has been repeatedly said, hyperæmia does not of itself indicate—it need not even suggest—pneumonia. Active pulmonary congestion owning a quite different origin may put on a precisely similar appearance, only to be distinguished from pneumonic engorgement by the help of collateral circumstances.

It is, however, not unusual to find, over a considerable tract in the vicinity of a hepatised area, that the pulmonary tissue is more intensely reddened and vascular and far less crepitant than elsewhere. Slight pressure upon the lung will cause the escape of frothy reddish serum from the spongy tissue, or this may even be manifestly œdematous and gelatinous-looking. Microscopically, the capillaries will be found to be distended and tortuous, and the alveoli to be filled with a clear fluid. Nevertheless it is not possible to declare that in these appearances we have the early stage of the pneumonic process; they may only indicate a condition of 'inflammatory œdema' around the hepatised lung. Or they may be the effect of the conditions of stasis obtaining at the time of death. Thus this state of vascular engorgement cannot, with certainty, be regarded as a pneumonic manifestation unless it be found elsewhere than in the dependent parts of the lung; for it is almost invariably present in the lower and posterior regions, not only in pneumonia but in all acutely febrile and exhausting diseases, being due to the cardiac enfeeblement, which is the primary cause of death in such cases, of which 'hypostatic congestion' and 'œdema' are acknowledged results.[1]

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[1] It is true that in cases of pneumonia one may find the non-hepatised lung in a state of œdema, sometimes in the upper lobe. Thus

But even when such hyperæmia is confined to the anterior part or apex of a lung, as when the other lung is already consolidated, it is still only conjectural that it is the first stage of the pneumonic process that is thus exhibited. Moreover it is to be inferred from clinical (physical) signs that this period of the affection is remarkably rapid in its evolution, passing sometimes within a few hours into the succeeding stage of red hepatisation, as in a case from which the accompanying figure was taken (Fig. 1). Even here, however, it is probable that the transition occurs not over the whole lobe at once, but by gradual progression. It may be inferred also—yet here again without much positive proof—that the hyperæmia may be so intense as to lead to hæmorrhage, for scattered points of hæmorrhage may be visible in the engorged tissue.[1] The congestion must be of the 'active' kind—that is, depending on vaso-motor paralysis rather than on obstructed venous outflow, and yet together with this there must also be an increased tension in the blood vessels : otherwise it is difficult to explain the phenomena that ensue.[2] The simplest explanation is that it is the first stage of an inflammatory process, modified, as it must be, by the special and peculiar conditions of the intrapulmonary circulation. The question whether the bronchial or pulmonary system of vessels takes the greater share in the process, is undetermined, but it is at least probable that the

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in the case of a lad, æt. 18, the following were the chief conditions noted : 'Recent pleurisy (right). Old adhesions over left lower lobe ; *recent pleurisy at apex.* Engorgement and red hepatisation of whole of right lung. Left lung : *œdema and engorgement of upper lobe* ; partial collapse of lower lobe.' ('Middlesex Hospital Pathological Report,' 1885, No. 173).

[1] The sputa are sometimes markedly sanguinolent.

[2] Professor Hamilton of Aberdeen has argued that 'croupous' pneumonia is really the outcome of 'undue blood-pressure suddenly applied,' and contrasts it with catarrhal pneumonia, to which alone he would grant the term inflammation. ('Pathology of Bronchitis,' 1883, p. 128.) Sir Andrew Clark has expressed somewhat similar views on the nature of pneumonia ('Med. Soc. Proc.' vol. viii., 1885, p. 103).

former play no inconsiderable part, since, as will be seen, the bronchioles themselves share in the later manifestations.

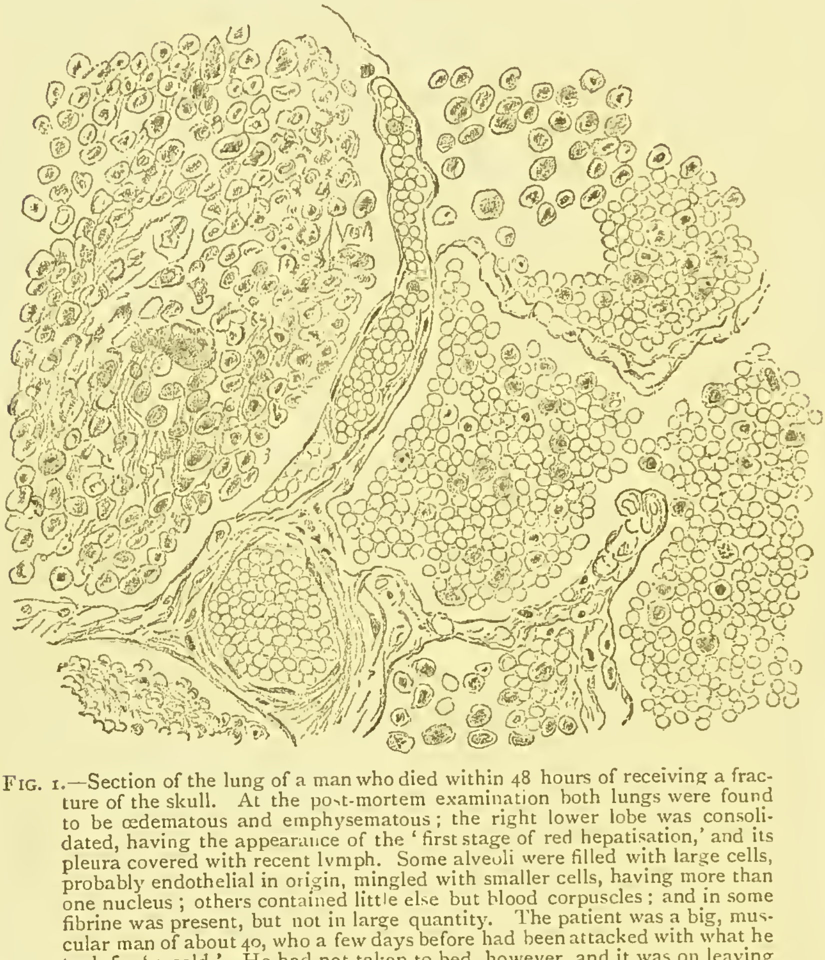

FIG. 1.—Section of the lung of a man who died within 48 hours of receiving a fracture of the skull. At the post-mortem examination both lungs were found to be œdematous and emphysematous; the right lower lobe was consolidated, having the appearance of the 'first stage of red hepatisation,' and its pleura covered with recent lymph. Some alveoli were filled with large cells, probably endothelial in origin, mingled with smaller cells, having more than one nucleus; others contained little else but blood corpuscles; and in some fibrine was present, but not in large quantity. The patient was a big, muscular man of about 40, who a few days before had been attacked with what he took for 'a cold.' He had not taken to bed, however, and it was on leaving a political meeting that by some accident, not fully explained, he fell headlong down a flight of stairs. (From a specimen prepared by Dr. Hebb, Pathologist to the Westminster Hospital.) (× 250.)

Stage of Exudation.—Engorgement is more or less rapidly succeeded by consolidation, in which all the air is expelled from the pulmonary alveoli by an exudation that coagulates within them. The result of this is a striking change in the physical characters of the organ, in bulk, weight, colour, and consistence, long since compared to the solid liver. The condition appears in two well-marked phases, alike as to their solidity, but differing markedly in colour and in histological characters. They are known respectively as (*a*) *red*, and (*b*) *grey hepatisation*, the former preceding the latter in the evolution of the disease, and the two generally spoken of as distinct stages.

Red Hepatisation.—The affected lobe presents a notable increase in bulk. It seems distended to the full, and as this distension is due to excessive fulness of its blood vessels and solid fibrinous exudate in the air-cells, the whole weight of the organ is *pro tanto* increased. When seen on removal from the body the hepatised lobe can be at once detected. It retains the shape of a fully-inflated lobe, its posterior border being widely convex, its diaphragmatic (we take the lower lobe as an example) surface somewhat concave, and its inner or mediastinal surface also hollowed to a certain extent. So bulky is it and so moulded to the chest wall that the portions which have been in contact with the parietes often exhibit furrowed depressions along the lines of contact with the unyielding ribs. These are rendered more obvious by the fact that the whole pleural surface is opaque and dull from inflammation, coated here and there, or in its whole extent, by flocculi and lamellæ of fibrinous exudation (recent lymph), beneath which the membrane is intensely injected and the seat of punctiform hæmorrhages. The rib-markings above mentioned are in such circumstances rendered more obvious, especially if the amount of lymph be small, for where they occur the pleura is plainly paler and less vascular than in other parts. The pleurisy extends over the whole of the consolidated lobe, so that the interlobar fissure is found to be occupied by lymph, and very often the pleuritic change may extend considerably beyond the limits of the pneumonic area, passing on to the contiguous lobe.

This lobe presents a marked contrast to the hepatised one. It is felt to be soft and crepitant, and it may be in part collapsed, in part over-distended with air (acute emphysema). The comparison between the two lobes is best made by means of a vertical section through the posterior border of the lung from apex to base, down to the root. The cut surface of the hepatised lobe presents a dark brick-red tint and a finely granular aspect. This 'granularity' is due to the solid fibrinous contents of the vesicles and infundibula bulging freely beyond the elastic alveolar walls. Actual casts or moulds of the infundibula, air-cells, and bronchioles may be obtained by scraping the cut-surface of the lung. These may be floated out in water and examined with a lens. There is no better proof of the coherent and coagulated character of the exuded material. It is as if the interstices of the spongy tissue of the lung had been filled with some injection-material which has become accurately moulded to them. The cut-surface is, however, dry, and no fluid can be expressed or scraped from it. The whole lobe is firm, solid, not very friable, but when broken its fractured surface shows the granular character very strikingiy.

These characters are fully explained on microscopical examination of sections of the hepatised organ. In the first place there is to be noted a marked engorgement of the vessels and pulmonary capillaries. The latter are more tortuous than natural, and bulge into the alveolar space, which is occupied by a mass of material composed of (1) red blood corpuscles in varying amount, sometimes so abundant as to suggest the idea of hæmorrhage rather than of mere diapedesis; (2) a few leucocytes; (3) many epithelioid cells, some in process of detachment from the alveolar wall, others mingled with the exudate in the cavity; and, most characteristic feature of all, (4) a fine meshwork of threads of fibrine, binding all these cellular elements together (Fig. 2). Often, indeed, the latter are so abundant as to entirely conceal this delicate fibrinous network, but where these elements are scanty it is well seen, or it may be brought into view by pencilling. It is the presence of this network that determines the pneumonic character of the specimen, and it has given rise to the terms 'fibrinous' and 'croupous' applied to this type of inflammation.

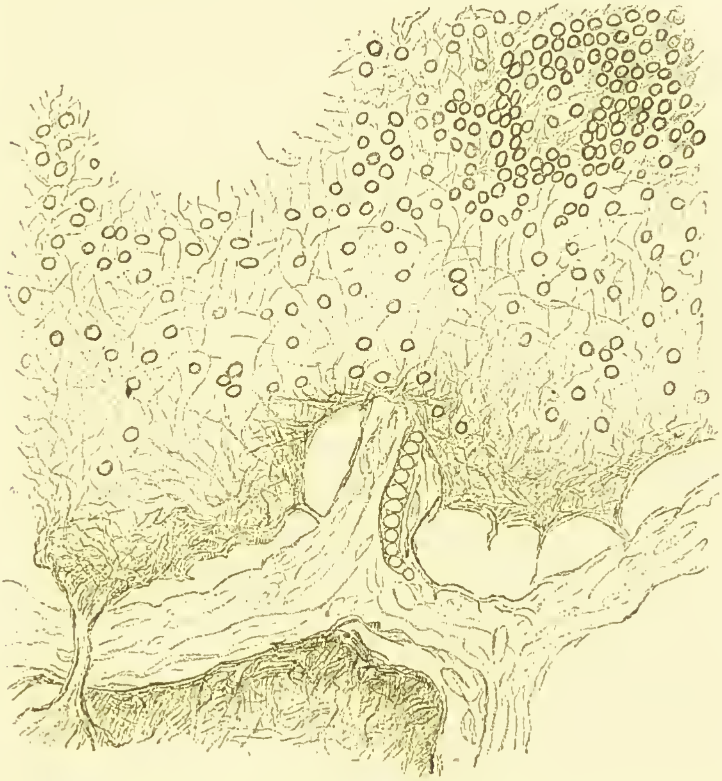

FIG. 2.—Section of lung in the stage of red hepatisation, showing an abundant and well-marked fibrinous meshwork within the alveoli, with some red corpuscles entangled in it. The specimen was obtained from a man æt. 42, who died after six days' illness. There was considerable chronic induration of the lung (chronic interstitial pneumonia), so that the alveolar walls are unduly thickened. There was also double chronic pleurisy. Both lungs were in a state of red hepatisation, except the upper third of the right lung, and had a consistency like that of cirrhotic liver. (From a specimen prepared by Dr. Hebb.) (× 250.)

Nor is the croupous exudation limited to the alveoli. It extends into the bronchioles, which it completely plugs. Nay, in some cases it even spreads into the larger tubes, forming solid cylindrical casts in bronchi of the third or second degree.[1] On removal of these plugs the mucous membrane will be found to be much injected or intensely reddened, a character which also obtains generally throughout the affected lung, apart from such croupous inflammation. There is always considerable muco-purulent or mere frothy sero-sanguinolent secretion in the bronchi.

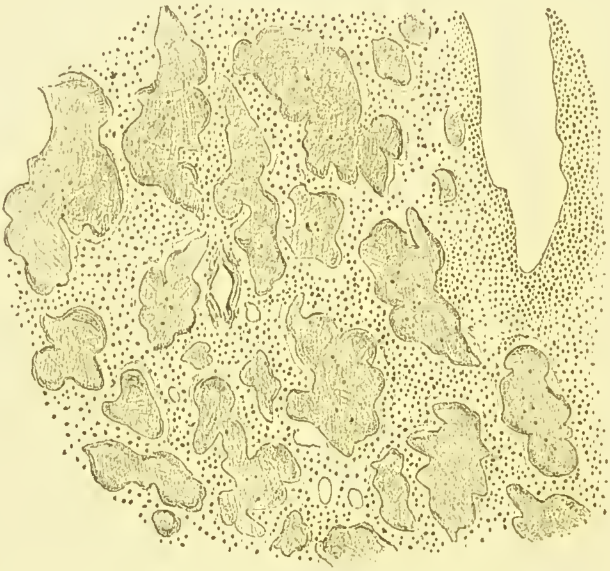

FIG. 3.—From the lung of a child attacked with 'lobar pneumonia.' To the naked eye the lung, which was almost completely consolidated and of firm section, had a granular surface on section. There was a thick layer of lymph on the pleura. The specimen shows, in addition to complete plugging of the alveoli with amorphous granular material (probably altered fibrine), a remarkable degree of small-celled infiltration of the alveolar walls, obviously of recent date. It seems to be an example of acute interstitial pneumonia combined with the ordinary 'croupous' type. (× 50.)

As a rule the alveolar wall shows but little alteration beyond the vascular engorgement, and if thickened the change is mostly of antecedent occurrence. But we have had the opportunity of examining a specimen, prepared by Dr. Hebb, from the case of a child affected with lobar pneumonia under the care of Dr. Leslie Ogilvie. The specimen shows (Figs. 3 and 4) a remarkable amount of leucocytal infiltration of these septa, whilst the lumina of the air cells are occupied with masses of granular fibrine. The specimen at first sight is suggestive of what has been met with in congenital syphilis, but not only is there no confirmatory evidence of that disease, but the interstitial inflammation is confined to the area of hepatisation, and, like the latter, is plainly of acute origin. The condition is, however, most exceptional.

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[1] Forming the 'massive pneumonia' of Grancher ('Gaz. Med.' 1878). In such cases the physical signs may simulate pleurisy with effusion, owing to the suppression of breath-sound from obstruction of the passage of air through the bronchi. (See Chap. IV, p. 55, note.)

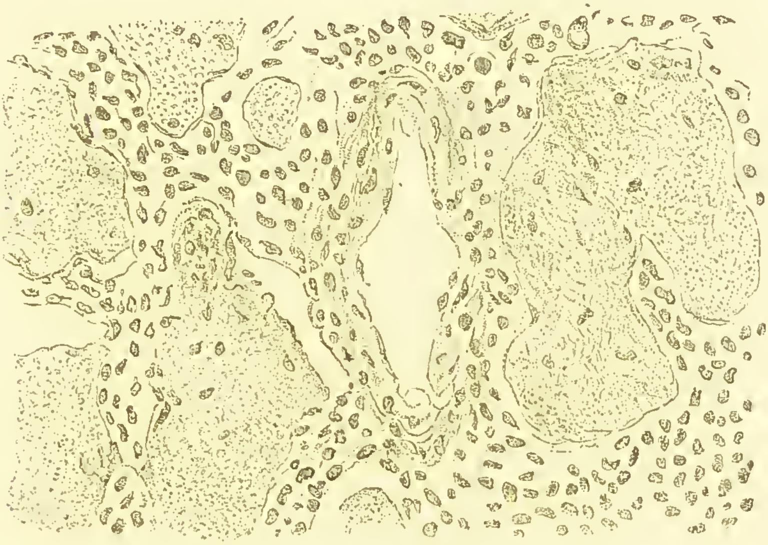

FIG. 4.—Shows a part of same specimen as Fig. 3, more highly magnified. (250 diams.)

Grey Hepatisation.—The characters of well-marked grey hepatisation present a striking contrast to those of red hepatisation. In many cases, it is true, we see transitional states, or rather observe that when one part of the lung is in the latter condition the contiguous portion is in the former, another evidence of the progressive character of the change.[1] In the state of grey hepatisation the lung still retains its greatly increased bulk; indeed, it may be more swollen than before, and the 'rib marks' more obvious. There is often considerable exudation of lymph upon the pleura, and generally some effusion into the sac, which may be clear and serous, or more often turbid, and occasionally quite purulent.

On section the consolidated lung has a greyish or yellowish-grey colour, is still markedly granular, but very friable. Scraping the cut surface yields a creamy fluid. Microscopically the alveoli appear distended with cell products. Their walls are distinct (Fig. 5), but the capillaries within them are no longer distended with blood, and are often wholly concealed by leucocytal infiltration. This comparative bloodlessness explains in some measure the pallor of the hepatised part, which

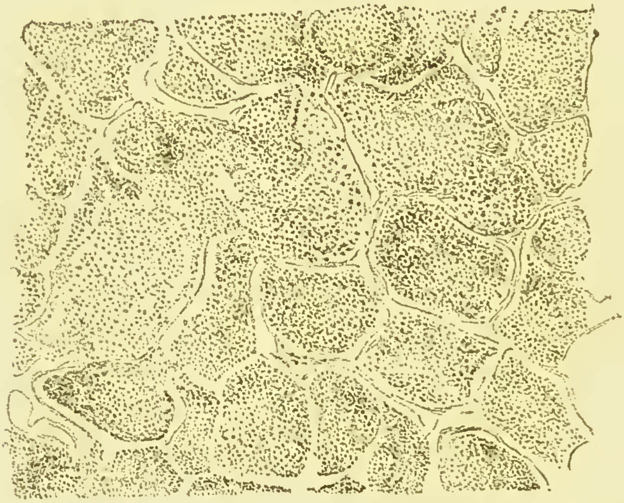

FIG. 5.—Grey hepatisation. (From a specimen prepared by Mr. L. Hudson, Pathologist to the Middlesex Hospital.) (× 50.)

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[1] The redness often seen at the base and posterior portions of a lung in grey hepatisation must not be mistaken for red hepatisation. It is due to the re-entry and accumulation of blood in the pulmonary vessels at the close of life (hypostasis).

is further augmented by the nature of the alveolar contents. These are now largely composed of cell forms resembling leucocytes and pus corpuscles, more or less filled with molecular fatty granules, mingled with larger nucleated cells derived from the alveolar epithelium, some containing granules of black pigment, and also with a quantity of amorphous material resulting from the disintegration of the fibrine observed in the earlier stage (Fig. 6). These appearances cannot be explained as the mere result of deranged blood pressure, they are evidence of the cell migration and proliferation, which stamp the change as being truly an inflammatory one, whilst also they denote the tendency to early degeneration of the cells and the transition between the preceding stage and the following one of liquefaction of the alveolar contents. The pigment,

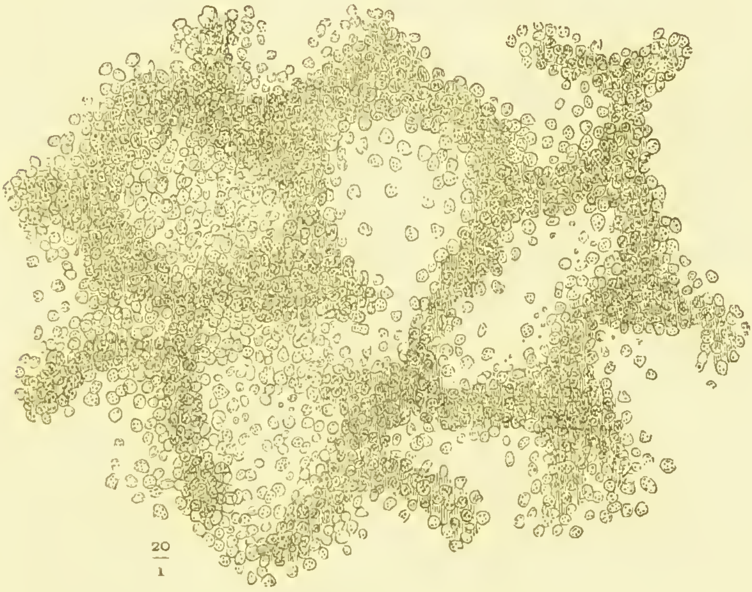

FIG. 6.—Pneumonia in the grey and softening stage, the alveolar walls exhibiting corpuscles similar to those they enclose, and in places showing signs of breaking down. (× 200 diams.)

partly contained in cells, partly in the form of free granules, is mainly derived from the colouring matter of the red corpuscles which occur in so large amount in red hepatisation. For in grey hepatisation it is hardly possible to detect any trace of red corpuscles ; they have, like the fibrine, undergone disintegration. The rapidity with which this degenerative change takes place in the exuded blood constituents is not the least remarkable feature of this disease. Although the alveolar wall is infiltrated with inflammatory corpuscles, yet the thickening is generally not marked, and rarely attains to the extent noted in the specimen above referred to (Figs. 3 and 4). Lastly, the bronchi may still retain some masses of fibrinous exudation, or muco-purulent secretion, besides being highly hyperæmic. The bronchial glands, too, are invariably swollen and inflamed.

Stage of Liquefaction.—The period of resolution of the inflammatory process is marked by the continuance of the degenerative changes already noted in grey hepatisation. The further mucoid and fatty degeneration of the abundant cell forms in the air vesicles converts the contents of the latter into a more or less fluid material, which is in small part expectorated, but in the main removed from the air vesicles by absorption into the lymphatics and veins. This stage is frequently presented to the morbid anatomist, since pneumonia is often fatal just at the period when liquefaction is commencing or fairly advanced. The cut surface of the affected lobe no longer exhibits a granular aspect, but is smooth, glistening, and very soft in consistence, often quite diffluent. It yields a copious milky juice on scraping (composed of fatty granules and degenerate cells). Often, indeed, one part of the lobe will exhibit these characters, whilst the rest will still have the features of grey hepatisation. The condition is often termed 'purulent infiltration,' a misnomer, for it implies rather an active process of suppuration than the merely passive one of degenerative melting down of the inflammatory products.[1] Under the microscope (Figs. 6 and 7) the alveoli are seen to be incompletely filled with granular amorphous material and opaque fluid, within which some shrivelled cell forms may be discerned.

Apart from this we do not possess any reliable evidence of the condition of the lung when the pneumonic process is advanced in resolution. We cannot observe the *restitutio ad integrum*. All we do know is that the lung may again become entirely permeable to air, and that functionally the organ becomes as competent as before the attack. Nor is there any evidence that in the vast majority of cases there is any perma-

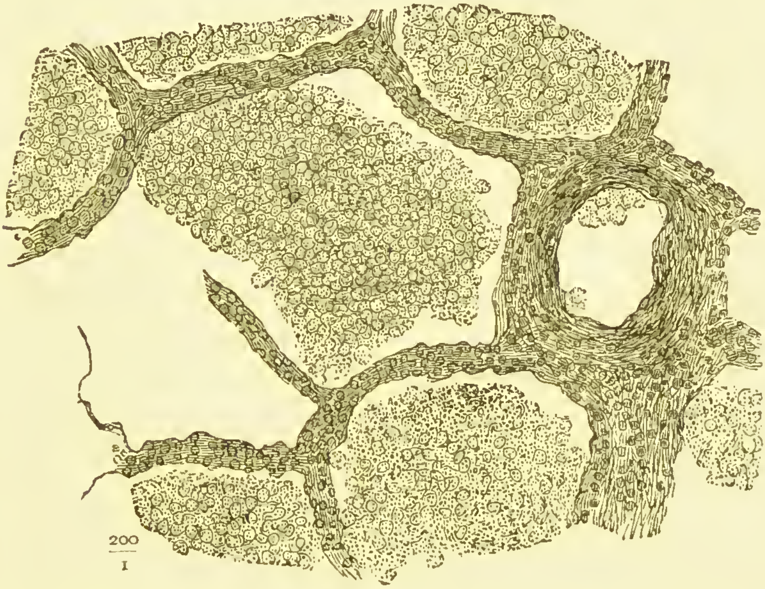

FIG. 7.—Hepatised lung in process of resolution. From a patient with pneumonia, dying when the physical signs of resolution had been established. The corpuscular elements lying loosely within the alveoli have undergone a granular conversion at the circumference.

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[1] This change is strictly comparable to the characters of softening cardiac thrombi, which, it is interesting to note, were once mistaken for suppuration.

nent alteration in its structure. It is true that we can only argue from clinical facts on the one hand, and from the characters of the lung in fatal cases on the other; but it is a pure assumption to assert that the changes (grey hepatisation, &c.) noted in the latter do not take place in the cases of recovery as some have ventured to maintain. The fact of such complete recovery seems to show that the inflammatory process is, after all, a *superficial* one, not involving the connective tissues, nor impairing the elasticity of the organ, and leading to neither abnormal dilatation nor collapse of the air-cells.

We may pause here to consider whether in a disease clinically so well-defined as pneumonia, it be not possible to assign correctly the special anatomical changes which accompany the various periods or stages of the disease, such as are denoted by the pyrexia and physical signs. It is undoubtedly true that this can be done to a certain extent, but a little consideration will show a lack of absolute parallelism between the anatomical and clinical features. At the outset, the latter denote the existence of a more or less widespread area of congestion in the lung, as well of incipient pleural inflammation, which are conditions of structural change possible to be detected in the dead body. Yet there must be a condition antecedent to these changes that constitute the so-called 'first stage,' for which there is no anatomical indication. Indeed, as we have seen, the stage of pulmonary engorgement has seldom been clearly demonstrated anatomically; for the obvious reason that at this early period of the affection death rarely occurs, and also because in cases which run the most rapidly fatal course, their very intensity carries them beyond this stage before death. And, as has been pointed out, it is extremely difficult to infer from post-mortem appearances the existence of ante-mortem congestion in any organ, and especially in an organ like the lung, which is so readily influenced by the circulatory changes that arise in the last hours of life, or in the act of dying. It is well known how at this time the blood tends to accumulate in the venous system, and to be especially prone to collect in the dependent parts from the failure of the heart to propel it onwards; and this resulting condition of hypostasis is even more liable to occur where the heart is already weakened by the febrile process. (See Chapter XI.) The lung may become so filled with sero-sanguinolent exudation as to be quite solidified, and the term 'hypostatic pneumonia' has been employed to denote this.

The next succeeding stage of acute pneumonia, of which there is clinical evidence, is that where consolidation has taken place, and where all the air has been driven out of the spongy interstices of the organ by the exudation from the blood. In some cases it is remarkable how early in the history of the attack the signs of such consolidation appear, following closely upon the preceding stage. And yet in this stage of consolidation there is manifestly clinical evidence, derived from the character of the expectoration, that it is divisible into at least two distinct phases, which, however, do *not* coincide with the two well-defined *anatomical* stages of red and grey hepatisation. The rusty, blood-stained, viscid sputa of the early days of an attack of pneumonia are, doubtless, in great measure due to the congestion that precedes consolidation, for it is plain that such products could not proceed from air-cells or bronchioles that are plugged with exudation. The continuance for some days of this type of expectoration goes far to suggest the fact (which often receives confirmation by physical examination of the chest, as well as by post-mortem inspection) that the pulmonary lobe is attacked not as a whole but *progressively* —the pneumonic process initiated at one part spreading thence with greater or less rapidity to another. Rusty expectoration may thus be inferred to continue so long as the local process is extending in the lung.[1] Its occurrence would denote the presence of the advancing hyperæmic zone, the precursor of hepatisation; whilst its cessation, or rather alteration in character, signifies the arrest of that extension. Hence the pathological stage of red hepatisation cannot be regarded as strictly conterminous with the period of rusty expectoration. The latter probably begins before the former, and only ceases when this has appeared at the last part of the lung to become involved.

What, then, is to be inferred from the change in the character of the sputa, when the viscidity gives place to fluidity, and the rusty colour to a greyish or muco-purulent secretion? Surely such change indicates the onset of resolution, not, be it again noted, over the whole of the affected area at once, but probably commencing at the part first to become hepatised, and thence spreading in the same order as was pursued by the course taken originally in the preceding stages. So here, too, the anatomical distinction between 'red' and 'grey' hepatisation does not accurately represent what is objectively shown during life. These two 'stages' are but parts of the same process, marked by the clinical signs of consolidation; and grey hepatisation is the immediate forerunner of resolution. That the later stages are more frequently met with after death depends on the fact that death usually takes place after the disease has existed for some time, but it is not uncommon to meet with each of the three stages present in different parts of the lung at the same time.

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[1] It is true that this type of expectoration, scanty, viscid, blood-stained, rusty, brownish, or orange-tinted, may be present quite to the fatal termination of a case, where after death grey hepatisation is the predominant feature. But in all such cases it will probably be found that there is also evidence of more recent change, elsewhere, in that or in the opposite lung.

We see, then, that morbid anatomy does, to a certain extent, confirm the notions derived from physical examination of the progress of the pulmonary lesion, but it does not wholly harmonise with the latter. It may, perhaps, mislead, from the fact that we are only dealing with a *fatal* case, if its teachings are followed too rigidly. For there is ample room for every conceivable modification (in cases yielding clinically the signs of pulmonary consolidation) in the appearances presented, not only as to the extent of lung involved, but as to the stage which may have been reached at the time of death.[1] Moreover, as cases differ in extent of the lesion, so do they vary in their intensity, and in the rapidity with which the changes are evolved, conditions which are fully exemplified in the morbid characters of the lung. Morbid anatomy affords further an explanation of the physical signs, and helps to explain anomalous deviations from their usual course. It is of great service also in informing us of the after-results of the pneumonic process, as well as in ascertaining the state of other organs which may have had a great influence in determining the fatal issue.

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[1] As illustrating the variability of the anatomical characters of the inflamed lung at the time of death, and the lack of any definite relation of these to the time at which death has occurred, the following cases may be quoted, where death took place at periods varying from 6 to 17 days from the onset.

1. Male, æt. 40. Death on 6th day. Grey hepatisation of greater part of left lower lobe.

2. Male, æt. 75. Death on 7th day. Grey hepatisation of anterior part of upper lobe (congested posteriorly) and of upper one-fourth of lower lobe of right lung. In left lower lobe a 'recent' pneumonic nodule.

3. Male, middle aged. Death on 7th day. Whole of lower and part of upper lobe of left lung hepatised ; *grey* at base, elsewhere *red*.

4. Male, æt. 44. Death on 7th day. Grey hepatisation and '*purulent infiltration*' of right upper lobe.

5. Male, æt. 62. Death on 8th day. 'Red passing into grey' hepatisation of right lower lobe.

6. Male, æt. 50. Death on 8th day. Grey hepatisation of right lung (except apex).

7. Male, æt. 24. Death on 9th day. Grey hepatisation of left upper and lower lobes. Patches of 'reddish-grey' hepatisation in right upper and lower lobes. (Fibrinous coagula in bronchi.)

8. Female, middle aged. Death on 9th day. 'Greyish-pink' (early grey ?) consolidation of right upper and lower lobes.

9. Male, æt. 48. Death on 10th day. Right lung, except extreme apex and anterior margin, entirely in state of grey hepatisation.

10. Female, æt. 41. Death on 10th day. Lower and part of upper lobe of right lung in state of 'reddish-grey' hepatisation.

11. Male, æt. 18. Death on 13th day. Both lower lobes 'dark-coloured and granular.' (This case was rheumatic, and was accompanied by peri- and endo-carditis.)

12. Male, æt. 50. Death on 16th day. Grey hepatisation, studded with yellowish points, of whole of right lung, except anterior and lower margins.

13. Male, æt. 49. Death on 17th day. Grey hepatisation of left lower lobe.

As regards the comparative frequency of implication of the two lungs, and of their lobes, many statistics are available.[1] The following are gathered from an analysis of a series of 144 consecutive post-mortem examinations at the Middlesex Hospital. In one of these cases there was no consolidation, but only a condition of general pulmonary engorgement, and, therefore, it was impossible to assert that it was really an example of pneumonia, to which, however, the clinical history pointed.[2] Of the remaining 143 cases, the right lung was alone involved in no fewer than 80, the left in 47, whilst in 16 both lungs were attacked.[3]

The distribution of the consolidation (marked 'c' in Table) in the different lobes of the lungs may be tabulated as follows :

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[1] Far fuller statistics are to be found in the Report of the Collective Investigation Committee, based, however, on clinical evidence alone.

[2] This was the case of a woman, æt. 37, admitted under the care of Dr. Douglas Powell on August 6, and dying on the 8th. There was high fever (T. 104·6), some dulness, and weak bronchial breathing at the base of the left lung, with coarse crepitation. There was also albuminuria. At the post-mortem examination the lungs were found to be extremely engorged, their lower lobes scarcely crepitant ; spleen deeply congested ; blood fluid. There was also congestion of the agminate and solitary glands. Dr. J. K. Fowler ('M. H. Rep.' 1883), who made the examination, states that the case was probably one 'of pneumonia, in which the anatomical lesion had not time to develop.'

[3] Osler ('Canada Med. and Surg. Journ.' May 1885) in 100 cases found the right lung involved in 51, the left in 32, both in 17.

—	Right alone						Left alone			Both								
Right upper lobe .	c	c	c	—	—	c	—	—	—	c	c	—	—	—	c	c	c	—
Right middle lobe	—	c	—	—	c	c	—	—	—	—	—	c	—	—	—	—	c	c
Right lower lobe .	—	—	c	c	c	c	—	—	—	—	—	—	c	c	c	c	c	c
Left upper lobe .	—	—	—	—	—	—	c	c	—	—	c	—	—	c	—	c	—	—
Left lower lobe .	—	—	—	—	—	—	—	c	c	c	c	c	c	c	c	c	c	c
No. of cases . .	13	11	12	23	7	14	9	12	26	2	2	1	4	1	1	2	2	1
Total . .	80						47			16								

From this it appears that the inflammation was unilobar in 71 cases, in two or more lobes of the same lung in 56, and in both lungs in 16.[1]

Lastly, the anatomical conditions which, in many cases, were variously combined may be summarised thus: Red hepatisation is recorded in 33·1 per cent. of all the series; red 'passing into grey' hepatisation in 24·5 per cent.; grey hepatisation in 36·2 per cent., and 'purulent infiltration' in only 6·1 per cent.

Pneumonia in its ordinary and typical course does not, as we have seen, implicate the framework of the lung, and consequently it is seldom that any destructive change or permanent induration ensues from a condition essentially transient. And although in very rare instances the evidence of such further change seems to be plain, it can only be in consequence of some added morbid process, some new departure in the progress of the inflammation, that such change can arise.

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[1] Osler found the following :—*Right.*—Whole organ (except perhaps a narrow margin at apex and anterior border) in 17; lower lobe alone in 7; middle and lower in 3; middle and upper in 2; upper and lower in 3. *Left.*—Whole in 10; lower lobe in 16; upper in 6. *Double.*—Mostly both lower lobes; in 3 cases lower lobe of one lung and upper of other; in 3 the upper lobes of both; and in 1 the left was in a state of uniform red hepatisation, except anterior border, and the right in grey hepatisation, except still smaller portions of corresponding regions. Altogether in 39 instances a lower lobe was involved, in 19 an entire lung, and in 16 the upper lobe.

As this subject will naturally fall to be considered in the chapter dealing with the after history of pneumonia (Chap. VII.), we will limit our descriptions here to a brief statement of such anatomical conditions as have been believed to be related more or less directly with antecedent pneumonia, and spoken of usually as rare forms of its termination.

Pulmonary Abscess.—This is a most rare event in the after history of acute pneumonia. It was found in but one case out of 144 examined after death. The abscess was seated in the lower lobe of the right lung, and had set up pneumo-thorax by perforating the pleura. There were patches of red and grey hepatisation in the upper and middle lobes.[1] It is more common, however, to find in the softening stage minute scattered foci of a yellowish colour, and composed of pus corpuscles; but there is no ground for believing that these represent actual disintegration of the alveolar walls, or that they are to be reckoned as abscess any more than that this stage is correctly termed 'purulent infiltration.' A true pneumonic abscess is a circumscribed collection of pus which may become limited by a definite pyogenic membrane in the midst of hepatised tissue, but what is the precise cause of its formation is really unknown. The only other abscesses or suppurating foci met with in the lung are those which are embolic in origin as in pyæmia, or which arise from the inhalation into the bronchioles of septic material, which sets up a lobular pneumonia that passes into suppuration (*Schluck-pneumonie*, of the Germans).

Pulmonary Gangrene.—Although more frequent than abscess, yet gangrene is also a rare sequel to acute pneumonia. Indeed, its connection with the latter has been questioned by high authorities.[2] It was met with,—in the series of 144 fatal cases of pneumonia already referred to,—9 times, the seat of the gangrene being in 5 in the right upper lobe, 1 in the right middle lobe, in 1 in the right lower lobe, and in 2 in the left lower lobe. Laennec's distinction between circumscribed and diffuse gangrene is one which fairly marks the two types met with, but it is remarkable that in pneumonia, where one might rather expect to meet more often with the diffuse form, it is commonly the circumscribed that occurs. The distinction does not imply any difference in origin, but is simply descriptive. In either case the appearances are those of a more or less ragged and irregular cavity, filled with highly fetid, blackish, grumous contents, and depending into which are the discoloured shreds of sphacelated tissue. The circumscribed character depends largely upon the extent to which reactive inflammation has been set up around the gangrenous focus comparable to the zone of demarcation observed in gangrene in other parts.[1]

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[1] This was a case admitted into the Middlesex Hospital in 1873, the diagnosis being pleuro-pneumonia. We are unfortunately unable to give clinical details.

[2] See later, Chapter VII.

Caseation.—It is a moot point whether caseation is ever an outcome of croupous pneumonia, although there are instances in which this change has been noticed, and in which there has been no evidence of tubercle. The appearances exhibited are those of the conversion of the hepatised area into a yellowish, or whitish yellow, dry, opaque, finely granular, very friable mass, which microscopically exhibits little but amorphous granular and fatty matter, mingled with the shrivelled relics of cells.[2] It is permissible to conjecture that the liability to this unusual sequel is dependent upon the degree to which the alveolar epithelium has shared in the inflammatory process. This, as we have seen, is variable; but there are cases in which there is considerable accumulation of such proliferated cells, with comparatively little fibrinous exudation (a class of case to which Buhl gave the name of 'desquamative' pneumonia). Ziegler[1] suggests that such a termination may be due to the special properties of the exciting cause, and that it is more likely to occur in cases where the pulmonary parenchyma is notably infiltrated with cells, and in which the walls of the lymphatics and blood-vessels share more freely in the inflammatory process.

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[1] For a well-marked example, see a case reported in the 'Clinical Society's Transactions,' vol. xii., by Dr. Cayley. The duration had been five or six weeks. There was a circumscribed gangrenous cavity in the lower lobe of the lung, the rest of which was reddened, consolidated, and finely granular. The cavity had been opened during life.

[2] See case reported by the late Dr. Lanchester and Dr. Hobson ('Path. Trans.' xxxvi. p. 115), where a child presented symptoms suggestive of empyema. The left lung was found to be solid, caseous, and dry, and there was distinct evidence of its having arisen in fibrinous exudation. It is true that here, as in other recorded cases of caseous pneumonia, the affection was associated with tuberculosis, which, however, in this case appeared to be of more recent date than the pneumonia.

Chronic Pneumonia (*Cirrhosis of Lung*).—Of more importance is the question of the possible conversion of the inflammatory products into organised tissue, and the consequent fibroid thickening of the lung. It opens up the subject of the changes undergone in hepatised lungs in which 'resolution' is long delayed. All writers admit this possible eventuality, but this fibroid change may be often better regarded as due to a process grafted on the primary pneumonic lesion. Nevertheless, it is sometimes possible to trace the direct conversion of the pneumonic products into fibroid tissue.

Dr. Thomas Addison in particular may be said to have given form to the conception of a chronic lobar pneumonia, and the subject has been fully studied by Charcot,[2] who attributes such fibroid conversion to (1) slow resolution, (2) prolonged pneumonia, and (3) to relapsing pneumonia. He distinguishes anatomically between 'red induration' and 'grey induration,' terms indicative solely of the superficial resemblance to the two forms of hepatisation, but not intended to imply any real connection with them. At the same time it is interesting to note that the cases which present the 'red' form of induration, are those in which the period dating from the onset of the disease is comparatively short, viz. from three weeks to a month. When the morbid process has extended over a longer period the characters approach more and more to those of grey or 'slaty' induration, whilst at an intermediate period (from one month to six weeks) the characters may be rather those of 'yellow induration' (Hope), 'yellow hepatisation' (Lebert), the albuminous induration of Addison. The following are the characteristics of the best marked varieties as given by Charcot.

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[1] Ziegler, 'Lehrbuch, Allg. u. Spec. Path. Anat.' 5th ed. 1887, ii. p. 658.

[2] Charcot, 'De la Pneumonie Chronique, Thèse,' 1860, and 'Œuvres Complètes,' tome v., 1888.

Red Induration.—The tissue of the affected lobe is compact, dense, non-crepitant, its cut surface dry and granular; the pleura is greatly thickened, but the bronchi not dilated. The alveolar walls are thickened by round and fusiform cells, sometimes by fibrillar tissue, the alveolar spaces narrowed, and filled with masses of epithelioid cells, contained in a granulo-fatty medium, in which margarin crystals may often be present.

Grey or Slaty Induration is described as existing under two distinct types. (1) The lung firm, dry, contracting, but still having a granular appearance on section; the bronchi not dilated, but the interlobular septa thickened. (2) Of longer duration, four or five months to a year or more, firm, 'crying' under the knife, slaty, greenish or blackish, smooth, slightly spongy, the pleura very thick, the lung contracted, but no dilatation of bronchi. The alveoli are diminished in number and narrowed, their thick fibrous walls showing polypoid ingrowths into the lumina. Cavities may form in these cases.

It must be admitted that such changes are rare, and indeed resolution, although sometimes much delayed, in the majority of cases appears to be complete after a time.[1] We cannot recall ever seeing a specimen which presented the characters of the above-mentioned 'red induration,' the nearest approach to the condition being that observed in Dr. Cayley's case of gangrene above alluded to, where the tissue surrounding the gangrenous cavity had the aspect of deep red consolidation, very tough, but smooth on section. We have, however, occasionally met with cases in which there have existed more or less extensive areas of slaty induration, but in comparatively few has there been any definite history of previous acute pneumonia. The best marked example was in the case of a plumber, the subject of chronic Bright's disease, who was in the Middlesex Hospital in 1878, under the care of the late Dr. Greenhow. The case is fully reported in the Pathological Society's 'Transactions,' vol. xxx., 1879, but as it is of special interest, we may venture to recall it here.

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[1] See Chapter VII.

The man had been ill for two months, but for some years had been subject to winter cough, and his lungs were very emphysematous. On the right side the pleural cavity was obliterated by adhesions in its posterior half, and there were other adhesions elsewhere, whilst the anterior part over the lower lobe was the seat of recent pleurisy. The posterior and lower part of the upper lobe was perfectly consolidated, the consolidation reaching upwards to within two inches of the apex of the lung, and corresponding mainly to the region of adherent pleura. On section, the consolidated part was found to extend inwards to the depth of about three inches; it was extremely firm and tough, 'creaking' under the knife, the cut surface presenting a uniformly smooth, glistening appearance, of a slate-grey colour variegated by scattered deposits of black pigment. Although the over-lying pleura was considerably thickened, there were no well-marked fibrous bands traversing the altered tissue; but an appreciable amount of thickening occurred around the bronchi and blood-vessels, which appeared on the cut surface as scattered white fibrous streaks. This indurated tissue was sharply marked off from the emphysematous parenchyma in front of it, the prominent margin of the former being very uneven in outline. Lastly, the pleura covering the lower half of the anterior part of the non-consolidated portion was thick and opaque.

With the exception of some cavities, presently to be described, the lower lobe was consolidated throughout, very tough and giving much resistance to the knife ; the lobe presented on section a smooth and glistening surface, which close inspection proved not to be quite uniform, islets of spongy tissue being perceptible here and there. It was of a greyish and, in parts, of a pinkish-grey tint, stippled with points of black pigment, not markedly traversed by definite fibrous bands, save those forming the walls of the bronchi and blood-vessels. The blending of the pleural layers above mentioned formed a tough, fibrous investment to the posterior border of the lobe, as well as to its inferior surface, which it had been found impossible to detach from the diaphragm. The upper and posterior part of the lobe was, however, the seat of a vomica, the size of a duck's egg, and of a like shape ; lined by a tolerably thick and definite pyogenic membrane, it contained a quantity of dirty, brownish-grey, semifluid material of highly offensive odour, whilst shreds of sloughing tissue depended at one part from the wall ; this wall was not smooth, but was rendered irregular and uneven by ridges running in various directions, as in a phthisical vomica. At the upper part of the cavity some further ulcerative action appeared to be in progress, for the cavity extended a short distance upwards into the adjacent portion of the consolidated upper lobe, the interior of this (more recent) part of the cavity being destitute of a definite lining and presenting a reddish colour. The cavity came close to the surface of the lobe above and posteriorly.

In addition to this, the posterior part of the lobe was further the seat of numerous small suppurating foci, none larger than peas, full of pus, and closely packed together, so as to give quite a riddled appearance to this part of the lobe. The tissue surrounding them, although as dense, firm, and smooth as the rest of the lobe, was of a dark, brick-red colour, so that it had a 'carnified' appearance, which, on examination with a lens, proved to be due to the minute vascular injection of the tissue.

The bronchial tubes throughout the lung were full of muco-purulent secretion ; their lining membrane was intensely reddened. It was thought that their calibre was slightly but uniformly dilated ; yet the dilatation, if any, was but small, certainly not proportionate to the extent and degree of the induration. At the same time the whole volume of the lower lobe was certainly reduced in size, especially when compared with the size of the upper lobe. The lung, with adherent pleura, weighed 48 oz.

Microscopically, under a low power the thickened pleura was observed to send processes into the pulmonary tissue, and the interlobular septa were obviously thickened and pigmented. Much dense fibrous tissue encircled bronchi and blood-vessels, the former exhibiting patches of black pigment in the midst of the more or less concentrically arranged bundles of connective tissue. The alveolar parenchyma presented a most intricate feltwork ; for although the outlines of the alveolar walls could be plainly made out, yet the alveoli were shrivelled, and their interior appeared to be filled with interlacing bundles of connective tissue running in all directions. Under a higher power it was remarkable to observe how in many parts the alveolar wall retained almost its normal tenuity. Its capillary plexuses were very prominent, but in the regions under consideration were hardly at all concealed by any thickening of the wall. And this occurred in parts where the interior of the alveolus was largely occupied by what appeared to be fairly well-developed fibrous tissue, not arranged in any definite manner, but forming coarse felted bundles, which passed at many points to join similar bundles in the neighbouring alveolus. A few leucocytes were here and there entangled in the meshes of this felted tissue, and, in some alveoli, cells of a distinctly epithelial type intervened between the capillary plexus of the alveolar wall and the organised contents. At the same time it was obvious that, in places, actual organic connection had occurred between the alveolar wall and the contents, for capillaries could be seen, filled with blood, here and there traversing the new fibrillated tissue. But that all these contents could not have been formed by outgrowths from the alveolar wall was shown by the study of other parts of the affected lung. Nor in all parts of the induration did the alveolar wall remain intact. Side by side with alveoli with unchanged walls were others where considerable thickening had taken place. Hardly anywhere, however, was it possible to fail in distinguishing the limits of the alveoli.

In close contiguity to advanced alterations like these were others showing transitional changes. There were parts, recently inflamed, where the alveoli were filled with a delicate meshwork of fibrinous exudation entangling leucocytes and catarrhal cells—appearances precisely those yielded by a recently hepatised lung. Again, other alveoli were filled with granular amorphous masses, evidently due to disintegration of the inflammatory products. Next in order came other alveoli, the walls of which were unaltered, but which contained within them a delicate, almost homogeneous substance, which presented distinct fibrillation, and which passed over at various points from one alveolus to another. In the interior of this material long and delicate spindle-cells could be made out, together with other bodies resembling leucocytes. One of the specimens exhibited showed this in a very striking manner (see Fig. 8). It showed the first stage of *organisation of the exudation products* of croupous pneumonia. From this to a more organised, fibrillated, and vascularised intra-alveolar meshwork there seemed to be all gradations of change. Fine and delicate capillaries containing blood could, here and there, be seen permeating the new tissue, indicating that organic connection had been made between the alveolar walls and the alveolar contents. Thus there was here evidence of actual organisation taking place in inflammatory exudation, probably through the medium of the exuded leucocytes. Certainly the appearances admitted far more readily of this interpretation than of that which assumed any ingrowth of

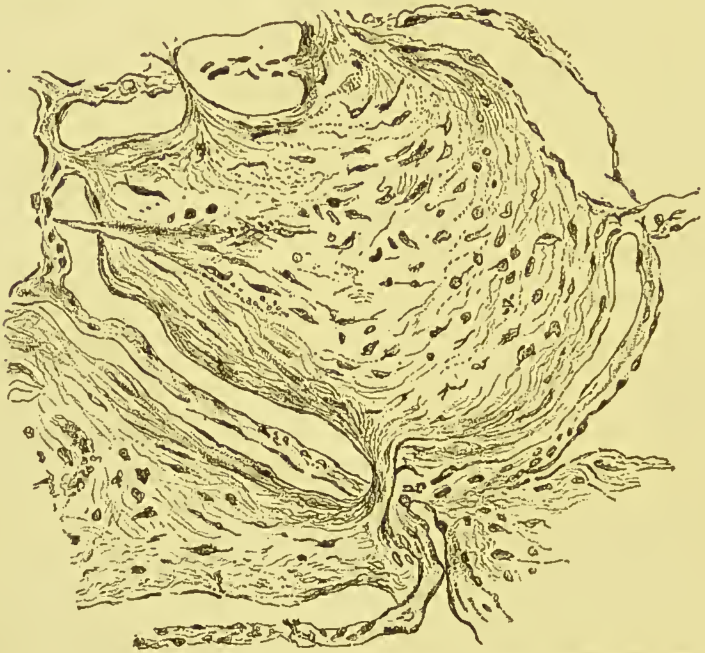

FIG. 8.—Portion of the consolidated tissue from the seat of 'grey induration.' The alveolar walls are almost unaltered, but the intra-alveolar exudation products show apparently the direct organisation of the inflammatory material in the development of spindle-cells and fibrillation. (× 500 diams.)

new tissue from the alveolar wall. Elsewhere there was seen the final fusing of the whole parenchyma into the nucleated fibous meshwork, the alveoli shrinking in bulk, and some of them presenting quite a shrivelled appearance.

There were also certain secondary changes in this lung which are not infrequent in chronic pneumonia. Thus there was a gangrenous cavity in the upper lobe, and scattered foci of lobular suppuration in the midst of the consolidated lower lobe, which had led in these parts to the destruction of the new-formed intra-alveolar tissue. It was surmised that this secondary suppuration had been caused by the gravitation of some of the fetid contents of the gangrenous cavity into the bronchi of the lower lobe.

We have dwelt at some length on the description of this specimen because it illustrates a fact, denied by some, that the pneumonic products may themselves undergo organisation. No doubt they are aided therein by the formation of vascular outgrowths from the alveolar wall, as has been so well shown and illustrated by Dr. T. H. Green,[1] but the actual conversion of the exudation into organised tissue has also been described by Marchand and others,[2] and cannot admit of doubt.

In process of time, doubtless, this condition of slaty induration would lead to a shrinking of the bulk of the lung, a great compensatory thickening of the pleura and dilatation of bronchi, the features, in fact, of some cases of so-called fibroid phthisis, but still all these changes have been initiated by an acute pneumonia, in which tubercular processes have had no share. The condition differs also from interstitial pneumonia, such as is produced by the inhalation of dust, or by chronic pleurisy, or in association with chronic bronchitis, not only in the clinical history but in the manifestly different mode in which the fibrotic process is evolved. It would take us beyond our limits to enter into the subject of chronic interstitial pneumonia ; and we must content ourselves with drawing attention to the fact that the 'fibroid lung' must be reckoned as one of the eventualities of ordinary croupous pneumonia.

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[1] Green, 'Introduction to Pathology and Morbid Anatomy,' 3rd ed. 1875, p. 347, Fig. 100.

[2] Marchand, 'Virchow's Archiv.,' lxxxii. 317. See also an interesting paper by Dr. Percy Kidd, entitled Subacute Induration Pneumonia, 'Lancet,' 1890, i. p. 740. In this paper will be found references to recent literature on the subject, and the conclusion arrived at by Dr. Kidd agrees with that of Heitler, in that these cases belong to a class distinct from ordinary acute pneumonia. Another paper on the same subject by Dr. Auld appears in the 'Lancet,' 1890, i. p. 792. The authorities quoted by Dr. Kidd are: Wagner, 'Deutsch. Arch. f. klin. Med.' Bd. xxxiii.; Heitler, 'Wiener Med. Wochenschr.' 1884 and 1885; Talma, 'Zeitschr. f. klin. Med.' Bd. x.; Hanan, *ibid.*, Bd. xii.; Pal, 'Fortschr. d. Med.' 1888.

Lobular Pneumonia.—A brief digression may here be made to speak of the essential anatomical characters of lobular or, as it is best termed, broncho-pneumonia ; not that this affection has much in common with lobar pneumonia, but in order that, by contrast, the features of the latter may be the more clearly brought into view. Lobular pneumonia is, in the vast majority of cases, if not in all, a *secondary* affection. It arises commonly in direct relation to acute bronchitis in children, often in the course of acute specific diseases, especially such as are complicated by bronchitis, *e.g.* measles, whooping-cough, and diphtheria. In the last-named disease, as in many other conditions, there can be little doubt that the pulmonary lesion is excited by the inhalation of foreign or septic matters into the bronchioles, and in such cases suppuration is the inevitable final result. The invariable occurrence of broncho-pneumonic processes in tubercular disease of the lung is also probably owing to a like mechanism, the bacilli of tubercle being in that case the foreign element that excites the inflammatory process. But of such cases we need not here speak, nor of those where embolism, septic or simple, sets up lobular inflammation.

In simple broncho-pneumonia of children the affection is almost always bilateral. It has been assumed, rather than actually demonstrated, that the inflammation ensues upon lobular collapse, and certainly it is common to find the two conditions side by side in these cases. This, however, only proves that both collapse and pneumonia may be caused by the same underlying condition, viz. the bronchitis, which, by causing obstruction of the bronchioles, favours collapse, and by extension of the catarrhal process into the alveoli produces pneumonia. In early stages the appearances are somewhat similar to those of red hepatisation, broken up as it were into several small areas, with intervening air-containing, but highly congested, tissue. The solid parts form nodular masses, which may be felt, or seen projecting above the surface of the lobe, and on section they project somewhat above the rest of the cut surface. They often have a finely granular aspect, but it would seem that this depends mainly upon the amount of fibrinous exudation contained in the alveoli of the affected area. For it cannot be too clearly stated that exudation of that kind is *not* limited to the lobar form of inflammation. At the same time there is, in lobular pneumonia, a far greater tendency to epithelial proliferation than in lobar. It is not rare to find the catarrhal and croupous characters combined in the same broncho-pneumonic focus, fibrinous exudation predominating in some alveoli, epithelial proliferation in others, yet the catarrhal character is that which is the most typical of broncho-pneumonia. In older foci it is usual to find the central part of each pneumonic focus paler and softer than the periphery, an appearance shown to be due either to puriform softening or to caseation, the latter process being favoured by the vast accumulation in the alveoli of epithelial products. In cases where the areas of broncho-pneumonia are closely aggregated, so closely as to simulate a lobar process, the detection of such centres of degeneration points to their lobular origin.[1]

The subject of the morbid anatomy of pneumonia is not exhausted by the record of the changes found in the lungs alone. The disease is associated in very many cases with inflammatory affections of other organs ; in varying proportion, it is true, but still often enough to suggest a considerable widening of the conception of the disease. Commonly regarded as complications, some of these lesions may seem almost as much parts of the pneumonic process as the lung affection itself. Since we have to speak of these in another chapter it will be unnecessary to dwell on them in this place.

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[1] This fact has led some observers to deny the existence of a lobar caseous pneumonia referred to above.

CHAPTER VI

COMPLICATIONS

Bronchitis—Emphysema—Collateral œdema—Pleurisy and empyema—Pericarditis—Myocardial softening—Endocarditis—Gastro-enteric disorders—Jaundice—Hepatic disease—Tonsillitis—Parotitis—Splenic and renal affections—Meningitis.

The course of acute pnuemonia is, in the majority of cases, unattended by any 'complications' in the strict sense of the term.[1] The disease may, indeed, be grafted upon pre-existing affections, and in this aspect we shall have to consider it hereafter ; but in speaking of complications, we are referring solely to the primary acute disease and not to its secondary or intercurrent forms. It is, in fact, material to discriminate between conditions which complicate pneumonia, and those which pneumonia itself complicates. Many chronic organic affections are terminated by the supervention of pneumonia. Yet, including all such cases, the statement of Huss,[2] based on 2,616 cases of pneumonia, was to the effect that only about one-third were complicated, the mortality in these being nearly four times as great as among the uncomplicated.

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[1] For the sake of convenience, we make a distinction between the complications of pneumonia discussed in this chapter, and its associations, which will be considered later on. By complications we understand certain local affections apt to appear sooner or later in the course of a pneumonia already recognised as such ; by associated diseases we mean affections such as acute rheumatism and delirium tremens, that sometimes concur with pneumonia and are apt to disguise its proper symptoms.

[2] Huss, 'Lungenentzundung,' Leipzig, 1861. An interesting table of the diseases complicating pneumonia is given by Townsend and Coolidge in their paper on the Mortality of Acute Lobar Pneumonia, 'Transactions of American Climatological Association,' June 1889.

Bronchitis[1] must be regarded as being, next to pleurisy, the most frequent complication of pneumonia. In many instances, indeed, there is evidence of slight bronchial catarrh ushering in the attack of the graver malady ; but there is no such direct connection traceable between the bronchial and pulmonary inflammation as subsists in the case of broncho-pneumonia. From one point of view, however, bronchitis may be regarded as an invariable concomitant, since the lining membrane of the bronchial tubes passing to a hepatised lung, or in the vicinity of the hepatisation, is found to be reddened, and the tubes to contain mucoid secretion or fibrinous exudation (*vide* Chapter V), but these conditions are essentially part of the local inflammatory process.[2] Bronchitis can only be considered as falling under the head of complications when, clinically, there are, in addition to the signs of pneumonic consolidation, rhonchi and râles scattered abundantly over both lungs. When severe, this bronchitis adds much to the gravity of the case, increasing notably the dyspnœa and cyanosis, and influencing the character of the cough and expectoration, the latter being more abundant and less viscid than in an uncomplicated case.

Sometimes, instead of complicating pneumonia, bronchitis may itself be complicated by the former affection. True croupous pneumonia, that is to say, may attack a bronchitic patient, may arise even intercurrently during acute bronchitis. And when, as so frequently happens, the bronchitis is associated with emphysema, it is likely that the physical signs of these states will completely mask those of the intercurrent inflammation, the existence of which may be only inferred from an access of fever, and great increase in dyspnœa and cyanosis.[1]

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[1] Grisolle says that about one-fourth of his cases of pneumonia were preceded by bronchitis.

[2] In a paper on Fibrinous Exudation into the Bronchial Tubes ('St. Barth. Hosp. Rep.' vol. xii.) Dr. de Havilland Hall refers to this condition as occurring in pneumonia, instances of which are recorded by Drs. Bristowe and Wilks in the 'Path. Soc. Trans.' vol. vi. Dr. Hall agrees with Dr. Wilks in the opinion that it is generally met with in asthenic cases. See *ante*, p. 76, and also p. 55, note.

Pulmonary *emphysema* does not afford immunity from pneumonia, as the case just cited shows, but the association can hardly be said to be common. It is, moreover, a very serious combination.[2] The occurrence of localised areas of 'acute' emphysema in the non-hepatised portions of lung may sometimes be met with, but the condition is of no material significance. It is an open question whether such compensatory emphysema does or does not contribute to the production of the sub-tympanitic (Skodaic) resonance elicited on percussion over the non-consolidated part of a pneumonic lung.[3]

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[1] As in a case recently observed (Mid. Hosp.) of a labourer 43 years of age, long the subject of chronic bronchitis and emphysema. He was admitted into hospital with a severe attack of acute bronchitis, T. 98·6; P. 76; R. 30. He was markedly cyanosed, the chest very emphysematous, sibilant rhonchi with prolonged expiration audible in all parts; a few subcrepitant râles at both bases. Expectoration scanty, mucoid, and viscid. Nine days after admission he was seized with pain in the right side, vomiting, increased dyspnœa (R. 58), and fever (T. 103·6; P. 140). The only addition to the physical signs which then developed, consisted in a notable increase in the amount of moist crepitation, with fine friction sound in the right axilla, but no dulness on percussion. Two days later the sputa were blood-stained. He became very delirious, and died exhausted on the fourth day of the presumed onset of pneumonia, and at the *post-mortem* examination the lower lobe of the right lung, covered with recent lymph, was in a state of red hepatisation; the rest of the lungs being œdematous and highly emphysematous. (It was noteworthy that this patient, who undoubtedly developed pneumonia in the hospital, was occupying a bed at the same end of the ward as did a very severe case of pneumonia admitted about the same time.)

[2] Huss gives a mortality of 23 per cent. in cases of pneumonia complicating emphysema. Townsend and Coolidge give 2 cases, 1 fatal.

[3] This note, which is constantly to be obtained, not only in pneumonia but also in pleural effusion and other conditions entailing compression or consolidation of a part of the lung, is by most writers referred to the 'relaxed' state of the pulmonary tissue in the unaffected portion, and apparently rather to collateral hyperæmia than to emphysema.

The condition of so-called '*collateral hyperæmia*,' or 'œdema,' of the lung opposite to that attacked by inflammation, must not pass without notice. It implies the engorgement of the vascular territory of the unaffected lung as a direct consequence of the interference with the circulation in the hepatised area, but it is doubtful whether so purely a mechanical explanation can suffice. The fact of such engorgement and œdema is proved both by clinical observation and morbid anatomy, but it may be remarked that it is seldom of any serious extent, save in the fatal cases, where a further cause for its supervention is to be found in the cardiac enfeeblement. Of course the possibility that the signs of congestion may be due to the extension of pneumonia to the other lung cannot be lost sight of.

Pleurisy is so common an accompaniment of lobar pneumonia as to constitute practically an essential part of the affection. It is present, in greater or less degree, in almost every case that comes to post-mortem examination, and not seldom it extends beyond the limits of the hepatised area, or involves also the opposite (non-pneumonic) side. Hence the old term 'pleuro-pneumonia' is a fairly exact description of the disease, although its use in human pathology is now mostly restricted to instances where the pleuritic signs are well marked, and the word might perhaps be usefully retained for cases with pleuritic effusion. It may, in truth, be said that pleurisy is only conceivably absent where the pneumonia is throughout its course 'central,' and never reaches the surface.[1] In the majority of instances the pleurisy is dry—*i.e.* fibrinous—and its presence is revealed clinically by pain and friction sounds, sometimes hardly to be discriminated from the fine pneumonic râle. In exceptional instances, where the pleurisy is extensive, and especially when the diaphragm is involved, the pain may be so acute and obstinate as to constitute a danger of its own by preventing sleep and causing a constant restlessness, which the ordinary remedies addressed to this symptom do little to relieve. In a larger proportion of cases, however, there is more or less effusion,[1] which, while it removes pleuritic pain, may persist after the resolution of the pneumonia and require paracentesis, so that convalescence is retarded. There would seem, in our experience, to be a tendency, in such cases, for the effusion to become purulent (*vide* Chapter VII).

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[1] Dr. Osler, Notes on the Morbid Anatomy of Pneumonia, 'Canada Medical and Surgical Journal,' May 1885, out of 100 cases of pneumonia found only 2 in which the 'pneumonia was deep-seated and did not reach the pleura.'

Empyema[2] is, therefore, at once a complication and a sequel of acute pneumonia. Its presence may be suspected when, after the crisis (which, in such cases, may be remarkably transient, and, except for the disappearance of rusty sputa, diminution in dyspnœa, and generally increased comfort of the patient, might be overlooked), there is persistence of dulness with bronchial breathing, at first suggestive of delayed resolution. Pyrexia may also continue, but become more remittent in type. Such signs, with the further evidence of effusion, derived from displacement of heart and enlargement of the side, justify exploratory puncture. It need hardly be said that the onset of purulent pleurisy, especially in children, must not be confounded with the onset of pneumonia.[3]

Pericarditis is an interesting and serious complication,[4] and in many instances its presence is not declared during life. It is mostly described as more common in association with pneumonia (and pleurisy) of the left side,[1] and hence to be regarded as due to direct extension of the inflammatory process to the pericardium. But an analysis of 144 post-mortem examinations (Middlesex Hospital, 1873 to 1889) shows that this is by no means the rule, for out of 24 cases in which acute pericarditis (lymph or pus) existed, in no fewer than 11 was the hepatisation limited to one or more lobes of the *right* lung, in 10 the *left* lung was involved, and in 3 both lungs. It may be remarked, however, that pericarditis was more frequently combined with pleurisy of the left side, since of these 24 cases, in 12 there was bilateral pleurisy, in 5 the pleurisy, like the pneumonia, was limited to the right side, in 4 to the left (in 3 cases the distribution of the pleurisy is doubtful).

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[1] Huss, *loc. cit.*, found 104 cases of fluid exudation remaining after complete resolution of the pneumonia (2,616 cases).

[2] Empyema was met with in 5 out of 144 fatal cases (Mid. Hosp.), and in 4 out of 123 non-fatal cases of pneumonia.

[3] Empyema as a sequel of pneumonia presents some peculiarities in early life, which will be noticed in the chapter devoted to Pneumonia in Children.

[4] In 3 out of 161 cases (Townsend and Coolidge), 2 of which were fatal. Of fatal cases: 5 out of 100 (Osler); 24 out of 144 (Middlesex Hospital P. M. Registers, 1873 to 1889); or in 31 out of 176 fatal cases (*ib.* 1867 to 1887 inclusive).

It should be noted that the proportion of pericarditis in these statistics is far higher than that usually stated, but it is right to add that frequently it was comparatively slight.[2] No doubt we have here a reason why this complication is so frequently overlooked during life, in addition to the fact of its signs being masked by, or confounded with, those of concomitant pleuritis.[3]

In the first edition of this work an analysis was given of 94 fatal cases of pneumonia, with respect to this question, and it is remarkable that in this series the association of *pericarditis* with hepatisation of the *right* lung was met with in 3 cases out of the 4 where that inflammation was present. In the fourth case the hepatisation was doubled, though the upper lobe of the *left* lung exhibited the most advanced stage of it.

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[1] This statement is made on the authority of Bouillaud.

[2] No case has been cited, however, in which there were not some shreds of lymph with subjacent injection of the serous membrane.

[3] Behier ('Conf. Clin. Med.' p. 274) quotes Ormerod ('Lancet,' 1852) as giving the following in an analysis of 1,410 cases of pericarditis:

Pneumonia, 117; with recent pericarditis, 19.
Pleurisy, 86; ,, ,, 6.
Pleuropneumonia, 62 ,, ,, 8.

Behier also states that Leudet met with 6 cases of pericarditis out of 83 pneumonias; Austin Flint, 8 out of 133; Behier himself, 3 out of 114. Huss in his 2,616 cases of pneumonia gives 22 of pericarditis, 12 of which were fatal cases.

Again, in 23 cases of recent pericarditis and hepatisation co-existing, extracted from the St. George's Hospital records, it was the *right* lung which suffered in 16, either solely (which was the rule) or with very slight participation on the part of the left lung. Of the remaining 7 cases 4 were not distinctive, that is to say, 1 exhibited extravasation of the right lung without pneumonia, in 2 the lower parts of *both lungs* were hepatised (the right most in one of them), and in the fourth case the upper lobe of the right lung was hepatised along with all the left. In none of these 4 cases was the pericarditis stated to be recent; in 2, at least, it was evidently of old standing. There remain 3 apparent exceptions. Two record hepatisation of the lower lobe of the left lung, together with a thin layer of recent lymph over the heart; the third had honeycombed lymph in the pericardium, along with hepatisation of the back of the left lung. From the situation of the consolidation, and still more from the *absence of pleurisy* in all these three cases, they would appear to have depended on hypostatic congestion rather than on true pneumonia.

The Tables analysed did not contain a single case of extensive hepatisation of the left lung only along with recent pericarditis, while they contained at least 10 in which that was the condition with respect to the right lung alone.

That the *muscular wall* of the heart suffers in the febrile process was insisted on by Stokes,[1] and this fact is advanced as one of the chief arguments in favour of antipyretic therapeutics. The high degree of fever characterising pneumonia may therefore be supposed to induce some cardiac degeneration, but the comparatively rapid course run by pneumonic fever is probably accountable for such changes being rarely observed. On the other hand, when from any cause there is already existing myccardial degeneration, when, that is, pneumonia attacks the subject of chronic heart disease, the existence of such cardiac disability adds greatly to the gravity of the case (see Chap. VIII, Prognosis). In either event, whether the heart failure be induced by the pyrexia, or be antecedent to it, the effect of such failure is to be seen in the increasing tendency to, and often fatal, engorgement of the uninflamed areas of lung.

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[1] See Diseases of the Heart and Aorta, Chapter VII, following observations by Laennec and Louis.

Acute Endocarditis—a more uncommon complication than acute pericarditis. Here, again, we may be permitted to appeal to the sure basis of anatomical fact, since the inferences drawn from physical signs alone may be deemed inconclusive. In 10 post-mortem examinations out of 144 (Mid. Hosp., January 1873 to September 1889) endocarditic vegetations were present (or about 7 per cent. ; pericarditis being present in 16 per cent.) ; but even in some of these cases the cardiac lesion had probably existed before the onset of the pneumonia. It is possible, also, that in a certain number a common source for the pulmonary and cardiac lesions existed in the rheumatic poison,[1] though this is certainly not true of all cases in which endocarditis is met with. Osler, in his series of 100 post-mortem examinations, found 16 complicated with endocarditis, 5 of the simple variety with warty vegetations, and 11 malignant or 'ulcerative.' The association of ulcerative endocarditis with pneumonia has been especially insisted on by Dr. Osler, and it has obtained additional significance since the discovery of micro-organisms in the latter disease (*vide* Chapter XVII). It is, however, remarkable that none of the ten cases above named belonged to the category of ulcerative endocarditis, which may be regarded as a sequela of pneumonia as much as a complication. Our own comparatively limited observation of ulcerative endocarditis does not tally with that of Dr. Osler. Nevertheless there is abundant testimony in support of this view, so that our experience must be regarded as exceptional. It must, however, be stated that in four out of the ten cases there was old disease, and that (as is the rule in ulcerative endocarditis) the recent vegetations were implanted upon one or other of the thickened valves. Such cases may possibly be deemed by some to be of the nature of ulcerative endocarditis, but they did not show the typical destructive lesions of the latter. Excluding these, and also one case in which there was a history of rheumatism eleven months previously, we are reduced to but five cases (or 3·5 per cent.) of acute endocarditis apart from previous valve mischief.

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[1] In one case of 'mitral and aortic vegetations' the patient had suffered from acute rheumatism eleven months previously.

Lastly, the lesions of *chronic endocarditis*—viz. thickening and fibrosis—were met with in 21 cases, in 17 apart from any recent change. This list includes 7 cases of well-marked mitral stenosis, which was the sole lesion in 4. In 5 others the mitral valve is described as 'thickened,' in 1 the aortic, in 4 both mitral and aortic, whilst in 4 more the valves generally are recorded as being thickened. These facts may be better appreciated when set forth in tabular form in relation to the lung which was the seat of hepatisation. As with pericarditis, so here, the association with right-sided pneumonia is comparatively large.[1] It is interesting also to note the relative frequency of double pneumonia in these cardiac cases.

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[1] These lesions occurred in 13 out of 80 cases of right pneumonia, or 16·2 per cent.; in 5 out of 47 cases of left pneumonia, or 10·6; and in 9 out of 17 cases of double pneumonia, or no less than 53 per cent.

TABLE SHOWING ASSOCIATION OF ENDOCARDITIS WITH PNEUMONIA.

—	Right Lung	Left Lung	Both Lungs	Total
Vegetations alone:				
Mitral	2	—	—	2
Aortic	1	—	1	2
Mitral and aortic	—	1	1	2
Thickening alone of—				
Mitral (stenosis)	1	1	2	4
Mitral (simple thickening)	3	1	1	5
Aortic	—	—	1	1
Mitral and aortic	4	—	—	4
Valves generally	1	1	1	3
Thickening and Vegetations:				
Mitral stenosis (veg. on M. and Tr.)	—	—	1	1
,, ,, (veg. on M. and A.)	—	1	—	1
,, ,, (A. thick.; veg. M.)	—	—	1	1
General thickening (veg. M. and Tr.)	1	—	—	1
	13	5	9	27

Of affections of the *digestive organs* there are few that can be strictly regarded as complications of the disease. We have already considered under Clinical History the occasional predominance of gastro-intestinal symptoms, such as vomiting, diarrhœa, and flatulent distension, either premonitory of the attack, or coincident with its onset, or arising later in the course of the disease. The occurrence of a 'critical' diarrhœa, indeed, has been recognised from antiquity. It may be concluded, however, that gastro-intestinal affections are not common as complications of pneumonia. Moreover, we have to rely almost entirely upon clinical evidence in this matter, morbid anatomy not showing any lesions of importance in the alimentary tract.

The complication of *jaundice*[1] is sometimes met with. It was noted during life in only two out of our previously quoted 123 patients. In one of these, which proved fatal, it was not only intense in character, but was associated with extreme cirrhosis of the liver. In the other it was comparatively slight and transient, and did not notably modify the course of the disease. The jaundice is not apparently dependent on duodenal catarrh, for there is no evidence of obstruction to the exit of bile, and the fact that, in a majority of instances, it is met with in right-sided pneumonia, has suggested some special local influence on the circulation in the liver, of which, however, the precise mechanism is unknown. In fact Behier observed it as often with pneumonia of the left side as of the right (viz. four times in each). We do not think it of any prognostic significance.

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[1] Grisolle met with jaundice in 20 out of 277 cases; Behier, in 8 out

Of morbid conditions of the *liver*, ascertained on post-mortem examination, by far the most frequent is fatty degeneration and infiltration. Thus it is noted in 45 out of 144 cases (in 4 combined with marked cirrhosis), or in a much larger number if the condition of 'cloudy swelling' be included under this head. For it is not easy in any individual case to determine the share in the hepatic change to be attributed to the pyrexial process, and what to the previous habits of the patient, a fatty liver being the commonest effect of alcoholic excess. Probably in all of the marked cases the latter factor has much to do with the lesion, just as it has in cirrhosis, which was met with in nine of this series of fatal cases. It may be remarked, however, that pneumonia is sometimes unexpectedly fatal in apparently healthy men given to alcohol, but not intemperate, and the event is explained post-mortem by the existence of fatty degeneration of the liver, not discoverable or suspected in life (see Case XII, Chap. IX). The condition of

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of 144 cases. The latter ('Conf. Clin. Med.' p. 237) noted this complication more with apical pneumonia than with basic.

chronic congestion (nutmeg liver) and induration, either as part of a general fibrotic change or in association with chronic heart disease, was noted in eight cases.

Tonsillitis is not a frequent complication of pneumonia. It may, however, occur at the onset of the disease or even as a precursory affection, especially in cases of 'sewer-gas' pneumonia. It may also arise as an intercurrent malady, but does not appreciably influence prognosis.

It is far otherwise with *parotitis*, which is either a concomitant or a sequel of pneumonia, and which is of grave significance. Some writers, indeed, look upon its supervention as of fatal augury; but this is not absolutely correct. It only occurs in markedly asthenic cases of the disease, *i.e.* either where the subject of the attack is already debilitated or when he becomes profoundly weakened by the severity of the attack. Thus it has been observed mostly in subjects over fifty years of age, although it is not limited to this period of life. As Grisolle says, it is 'nearly unknown in childhood and youth, very rare in the adult, and chiefly noticed after the age of sixty.'[1] It almost invariably passes on to suppuration, giving rise to the 'parotid bubo,' which is perhaps more familiar as a sequel to typhoid and typhus fevers, and in association with abdominal suppuration, than with the disease we are now considering. The occurrence of this complication is indicated by the appearance of a painful swelling behind the ramus of the jaw, a swelling which may attain considerable dimensions, and which rather rapidly suppurates. The pus is thick, and escapes with difficulty through the incision, which is early called for. Parotitis is, we believe, more often unilateral than bilateral. Its precise pathogeny is not clear. It would seem to be of septic nature, but that it is always indicative of a general septicæmia, analogous to pyæmic manifestations, is by no means certain. Recent investigations would point rather to local infection from the buccal cavity ; if that be so, it is surprising that it should not occur more often.[1] We have met with one instance (*vide* Chap. IX, Case XV) in a young man, in whom the pneumonia was of severe type, and who was for some days in a critical state, but who eventually made a good recovery.

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[1] Grisolle ('Pneumonie,' 2nd ed. p. 382) also says that parotitis occurs towards the period of resolution of the pneumonia and with the onset of convalescence. As a 'critical' phenomenon it is of ill omen. The affection, which is hardly ever bilateral, rapidly increases, and in most cases goes on to suppuration or gangrene. It is not only indicative of a grave condition, but is in itself a most serious complication. Behier (*loc. cit.* p. 203) speaks of parotitis as a rare and little known complication of pneumonia, but of fatal augury. He relates three cases personally known to him. In one, a man, æt. 63, suffering from right basic pneumonia, parotitis appeared on the left side on the fifth day, and death occurred two days later. In the second, a man, æt. 64, on the sixth day (apparently the day after crisis) there was recurrence of fever with swelling of the right parotid, which suppurated slightly; death took place in four days. The third patient was a man, æt. 74, admitted into hospital on the eighth day of a pneumonia, which was beginning to resolve, when both parotids became inflamed, and death took place five days afterwards. Behier speaks of the complication as indicating nearly 'inevitable death.'

Affections of the *spleen* complicating pneumonia are practically limited to the swelling and softening sometimes observed post-mortem, and rarely attaining such a degree as to be palpable during life. The condition is probably referable to the febrile process.

We come now to speak of *renal disease* in association with pneumonia, and in view of the great importance of the subject, especially as to diagnosis and prognosis, we propose to deal at somewhat greater length with this variety of complication than with those above described.

There is direct clinical evidence to prove that imperfect elimination of urea is to be reckoned among the causes which predispose to pneumonia. Defective secretion on the part of the kidney (whether from acute or chronic nephritis) may have this amongst other consequences. In rare cases, indeed, pneumonia and acute nephritis may exist together. To these we shall refer in a future chapter (Chap. XI). But it is to be observed that in those instances where, from the history, it seems probable that the latter disease is a consequence of the former, the pneumonia is not deprived by this association of those features which commonly distinguish it. According to Dr. Dickinson, the marked tendency to inflammation which characterises lardaceous disease of the kidney is seen most conspicuously in the lungs, and next often in the pleura. Tubal nephritis, on the contrary, is associated with pericarditis and pleurisy more often than with pneumonia, while in granular degeneration (where the commonest inflammatory affection is bronchitis) neither pleurisy nor pneumonia is often met with.[1] Of course, these statistics fail to show precisely the liability to pneumonia which these affections respectively confer. They require correction in reference to the fact that pneumonia is a disease of early adult life, and thus likely to concur (even when arising from independent causes) with the forms of kidney disease proper to that age; with tubal nephritis, that is, rather than with granular degeneration.

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[1] A. Hanau (Ziegler and Nauwerck's Beiträge, 'Path. Anat.' iv. p. 487) maintains that inflammations of the salivary glands complicating other diseases are of local origin. One of his five cases was acute pneumonia.

In 94 cases examined post-mortem, and described at the time as pneumonia, there are 14 which seem to have this relationship.[2] The hepatisation in each case is that of true pneumonia, confined to one lung, with boundary sharply defined, and the chief seat often the upper portion of the lung. In 7 the kidneys were granular, and in 7 large, white, mottled. But of the granular, all but 1 were over forty, and the eldest sixty-seven, while of the large white, all but 1 were under forty, the youngest a child of two, and of the remaining 4, 5 were under thirty. Pericarditis is mentioned twice, namely, in a patient of twenty-six with large white kidney, and in one of forty-five with early granular. The numbers are small and, of course, inconclusive for showing the relative frequency of pneumonia in the several forms of kidney disease. Other cases occur amongst the ninety-four where there is renal disease, but it is only in those quoted that the association with the lung inflammation can be fairly assumed.

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[1] See Dickinson, 'Diseases of the Kidneys,' Part II. p. 484.

[2] This enumeration and the cases quoted in illustration are supplied from St. George's Hospital, and were inserted in somewhat different form in the first edition of this work.

In examples of wasted and granular kidney the post-mortem appearances, so far as the lungs are concerned, are characteristic of acute pneumonia—extensive hepatisation that is, in parts grey, softened, and readily breaking down,—but the symptoms are little obvious, and attention is mainly called to a prostrate condition, which only physical examination of the lungs explains.

Of the seven examples of pneumonia in connection with wasted kidney cited above the following may be quoted :—

> A woman of forty-five was admitted in a prostrate state, from which she never rallied. There were no symptoms pointing directly to the lungs, and she lived but a few hours. The kidneys were shrunken and finely granular, with diminished cortex, the organs chiefly consisting of fibrous tissue. The greater part of the left lung was in a condition of diffuse hepatisation, exuding puriform fluid in many places. The lower lobe of the right lung was in a state of red hepatisation. There was recent lymph on the surface of the left lung. The heart was hypertrophied, and in the pericardial sac was much recent lymph, layers thereof in adhesion to each other.

In the following the persistence of vomiting might of itsel have given a clue to the renal disease.

> A woman of sixty-seven, wasted and worn and with characteristic aspect of advanced granular kidney, was admitted for general pains about limbs of no definite character. She had also severe pain at epigastrium and frequent vomiting. During the last eight months

she had been almost constantly sick. There was no cough nor spitting, nor marked pyrexia, and during her six days' residence vomiting was the main symptom. She gradually sank and died. At the post-mortem examination the kidneys were found to be granular, contracted, and cortex almost absent. The lower lobe of left lung was solid throughout, in parts red, in parts grey and breaking down. Lower lobe of right was also hepatised, but in earlier stage. There were some firm adhesions. Recent lymph covered the inflamed portion of lung.[1]

The extreme rapidity of fatal issue in cases of pneumonia dependent on renal disease is well shown in the following case:—

A labouring man, aged twenty-eight, had been ill five days, and during the last two had had cough, pleural 'stitch,' and some rusty expectoration. He had pyrexia and some dyspnœa, but not until the second day were physical signs of pneumonia of right apex detected. On the fifth day he became delirious and very violent, and obstinately refused food. Expectoration soon ceased and dyspnœa was not urgent. On the sixth day from admission (being the eleventh day from the time he left work) he became cold and collapsed, and so died. On post-mortem examination the kidneys were found to be large and of granular surface, the capsules thickened. Liver large but not otherwise altered. The right lung was completely hepatised and adherent, the upper lobe the more advanced, grey and breaking down, the rest red. The left lung was healthy.

Though so important for prognosis, it is not always easy to interpret aright the indications afforded by the urine in cases like the foregoing. Albuminuria, as we have seen, is of common occurrence in pneumonia. But before suspecting the kidneys of disease it is necessary to ascertain the precise character of this particular symptom—a task that may be difficult or impossible so long as the actual stage of inflammation endures. A large amount of albumen with the presence of epithelial casts—indications of tubal nephritis—are not necessarily signs of pre-existing renal disease, inasmuch as in rare cases, as we shall relate, both lung and kidney are inflamed together, and from a common cause. But far more commonly chronic nephritis is a predisposing cause of pneumonia, which will become evident on careful inquiry as to the patient's condition immediately antecedent to the pulmonary attack.

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[1] As indicating the supreme importance attached to physical signs apart from all else, the following example of forty years ago is of interest:—A man of fifty-seven, admitted to the surgical ward for rupia of six weeks' duration, was transferred two days after admission to the physician, as he was observed to be in a very depressed condition. He had dry tongue, difficult articulation, but no confusion of mind, and lay constantly on the right side. Owing to the physical signs indicating consolidation of the right lung, he was cupped to ten ounces, and afterwards dry cupped. He died the same day, having been three days only in hospital. The kidneys were highly granular, with diminished cortex and numerous cysts. The heart was hypertrophied, weighing fifteen ounces. The middle lobe of right lung was almost wholly consolidated and of light buff colour; bronchial tubes very vascular and full of bloody mucus; fluid but no lymph in pleura.

There is another source of error. Renal disease may be ascertained beyond question, and, along with general anasarca. auscultation may discover small crepitating râle. Let it be remembered that in such a case pneumonia is not proved without further evidence than that which is afforded by this sign. Œdema of the lungs, like œdema of the limbs, is very apt to overtake such subjects, and from likeness of sound, to be mistaken for active inflammation. The crepitation of pulmonary œdema may resemble very nearly that of commencing pneumonia. The occurrence of crepitating rhonchus is, of itself, as has been mentioned, of doubtful significance. When heard at the base of one or, much more, of both lungs in an œdematous patient, without consolidation signs or marked pyrexia, it will indicate not a new disease, but the extension to these organs of the general dropsy.

It is necessary, moreover, to separate from pneumonia the consolidation (to be described hereafter) which is apt to follow upon such œdema, and is associated with chronic heart and kidney affections. Insidious in origin and of indefinite duration, this hypostatic solidification is far more common than the acute disease for which it is often mistaken. This latter for the most part, as we have shown, attacks the subjects of kidney disease as it attacks others[1]; it does not steal upon them unobserved in the midst of their other troubles, or attach itself to an already carnified lung. Pneumonia having this source needs separate recognition for its special danger. Though, at first, it may seem to follow the usual course, yet when the time for crisis arrives the patient sometimes rapidly sinks, and the lung is found to be disorganised.

These cases are the more misleading as their immediate exciting cause may be some notable exposure such as is known to excite pneumonia in a previously healthy person, while moderate pyrexia, absence of pleuritic pain and of extreme dyspnœa, may seem to justify a favourable prognosis.[2]

Of complications connected with the nervous system the most prominent is undoubtedly *delirium tremens*, which sometimes, as will be shown, serves to mask the lung affection (*vide* Chap. XI). There can be little doubt that alcoholic excess, which, *per se*, favours the liability to pneumonia and adds to its gravity, is often a factor in many cases of apparently simple delirium. But as has been especially pointed out by Juergensen the occurrence of delirium is also influenced by another factor, namely, the rapidity of onset and degree of the pyrexia present. It may, however, we think, be truly stated that the liability to delirium in the alcoholic is less dependent upon this latter factor than it is in the non-alcoholic.[1] As regards delirium tremens, however, there can be no question as to where the relationship exists, and its onset is a grave complication.

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[1] It may be asked, what amount of kidney change observed after death is sufficient to prove that the pneumonia is consecutive? How is the distinction to be made between pneumonia arising directly from imperfection of the kidney, and the same disease arising independently in an individual possessing that imperfection. It is obviously impossible to make it, and for this reason, no less than from the difficulty already mentioned of appreciating at the time the exact bearing of albuminuria, statistics upon the subject are encompassed with difficulty and error.

[2] The following may be quoted as an example :—A cabdriver, aged fifty-eight, was admitted into Westminster Hospital with pneumonia following exposure when drunk. It was the seventh day from the rigor (temperature 101·2, pulse 108, respirations 48). During the following two days temperature fell to 99°, pulse to 100, respirations to 40. He died on the twelfth day of the disease, temperature in rapid descent some hours before. Post-mortem examination showed, together with granular degeneration of the kidneys, hepatisation of the lung without pleurisy.

The great nervous prostration, associated sometimes with crisis, especially in the aged, has been already mentioned. It is evinced not only in an increase of delirium, but sometimes in the development of acute mania (which may also arise during the height of the fever) or of melancholia (see Case VI, Chap. IX).[2]

From time to time there have been recorded cases of *meningitis* complicating acute pneumonia.[3] In some this lesion has been found in association with pericarditis, in others with endocarditis. The predominance of cerebral symptoms may, indeed, lead to an erroneous diagnosis, meningitis especially, as we shall presently see, in the case of children. Herein, no doubt, may be found an explanation of some instances in which recovery is said to have ensued in spite of this complication. For, with rare exceptions, it must be said that the supervention of meningitis upon pneumonia is lethal. Its presence may be masked by the fever and pulmonary signs, and such cases have been termed 'latent' meningitis; but in others, the development of severe headache, of cerebral irritation, of paralyses, oculo-motor, or facial, or even of hemiplegia, will reveal the true nature of the complication. Of late years the combination has been more widely studied, especially in relation to the discovery (widely confirmed) of the *diplococcus pneumoniæ* in the meningitic exudation.[1] More will be said on this point hereafter, but it may suffice here to note that such discoveries only confirm the surmise of Behier as to the secondary character of the meningeal inflammation.

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[1] It is generally admitted that delirium and other nervous symptoms are more frequent in apex pneumonias, and that is also the impression gained from our experience. But the matter has still to be subjected to accurate analysis, in regard not only to the localisation of the pulmonary inflammation, but also with respect to the severity or intensity of the febrile process.

[2] Greenfield, on Insanity as a sequel of acute Disease and Hæmorrhage, 'St. Thomas's Hospital Reports,' 1874.

[3] Grisolle avers to have found recent purulent 'arachnitis' in 8 cases of severe delirium. Behier (*loc. cit.*) gives 2 cases, and draws attention to the slowness of the pulse (84) in 1, in spite of high fever, as an important indication. He refers to 1 recorded by Andral ('Clin. Med.' ii. p. 444). Barth and Poulin ('Gaz. Hebd.' 1879) draw attention to the comparative frequency of the complication, and in a later paper ('Rev. de Méd.' 1882) the former details a case associated with endocarditis. Osler (*loc. cit.*) met with no fewer than 8 cases of meningitis in 100 post-mortems; in 5 of these there was ulcerative endocarditis, and he suggests that embolism was the nexus between the conditions.

Of wider bearing is the observed relationship of pneumonia to *cerebro-spinal meningitis*, whether epidemic or sporadic. So marked is this connection, that it may be questioned whether every case of meningitis complicating pneumonia should not be considered as falling under the head of infective disease. At any rate, the more the subject is studied, the rarity of simple as distinguished from infective meningitis becomes the more obvious. The points in favour of a special relationship between pneumonia and cerebro-spinal meningitis are: (1) the concurrence of the two diseases in epidemic form; (2) the supervention of one or other affection in the course of either during the prevalence of epidemic cerebro-spinal meningitis; and (3) the results of bacteriological research.[2]

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[1] See especially Netter ('Archives de Méd.' 1887, and 'La France Méd.' June, 1889).

[2] See Immermann and Heller ('Deutsch. Arch. Klin. Med.' v.), who give 9 cases of meningitis in 30 fatal cases of pneumonia, occurring during the prevalence of epidemic cerebro-spinal meningitis. A case which commenced with some symptoms of cerebro-spinal meningitis, but which on the third day gave way to signs of acute pneumonia, is related by Hun ('Albany Med. Annals,' Aug. 1888). This case, which recovered, is hardly conclusive. Not only is it remarkable for the apparent 'metastasis' above noted, but also for the equally remarkable rapidity with which the crisis ensued after the declared onset of pneumonia.

In conclusion, it will be seen that the complications of pneumonia are varied, though for the most part infrequent, if we detach from the category conditions pre-existent to the acute disease. But the association with acute inflammatory manifestations in other organs, is a fact of considerable importance, not only in the pathology of the disease, but also with regard to prognosis. On both these points we shall have opportunity to dwell in the sequel.

CHAPTER VII

THE AFTER-HISTORY OF PNEUMONIA

Liability to repetition—Delayed resolution—Chronic pneumonia—Abscess of lung—Gangrene—Relations to pulmonary phthisis.

THE clinical history of pneumonia may be prolonged beyond the limits commonly assigned to it. In some few cases, indeed, as we have already seen, changes take place in the lung which are the direct outcome of the inflammatory lesions. To these we shall presently return, but before doing so it may be well to consider one point of especial interest, namely, the alleged *liability of pneumonia to recur* in a subject who has been once attacked. If we were to judge by hospital experience alone, we might assert that this is a matter of popular faith rather than of medical observation. In any series of cases derived from hospital records, there are comparatively few where the attack, like that of acute rheumatism, is a second one; and, indeed, so far as we are able to judge, but few of those admitted subsequently become the subjects of pneumonia. It is, however, certain that the converse proposition is not true, viz.: that any 'protection' is afforded from subsequent attacks. Indeed, if pneumonia be regarded as merely an 'inflammation of the lungs,' what more natural than that there should be a liability to recurrence in the same part, the *locus minoris resistentiæ*; or on the other view of its nature, namely, that it is a general disease, with local manifestation in the lung

—a disease of the zymotic class—the experience of such affections might point, either, as in malaria, to such liability, or, as in scarlet fever, small-pox, &c., to distinct protection from recurrence.

In the returns made to the Collective Investigation Committee there was a previous history of pneumonia in 101 cases out of 967, a considerable proportion, far exceeding that of hospital practice. These figures are, however, based on returns from practitioners, who have enjoyed the opportunity of knowing more intimately than is possible to the hospital physician the previous state of health of their patients. In ten of these cases more than one prior attack is recorded. It must be further pointed out that no other disease shows an equal tendency to attack pneumonic subjects with pneumonia itself; the next in order being bronchitis, and the next rheumatism.[1] If, then, this statement be at all near the truth, notwithstanding, that is to say, the probability of some error in the statement or interpretation of past symptoms, it fully justifies the opinion that pneumonia does predispose to another attack.

Other testimony is to like effect. Grisolle found among 175 cases no fewer than 54, or about 30 per cent., who had had previous attacks, a proportion three times as large as that furnished by the 'Collective Investigation Report.' Some of these patients had had as many as eight previous attacks ; and even

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[1] A previous history of the following illnesses was returned in this series of 967 cases:

Pneumonia	101 cases	10·4 per cent.
Bronchitis	84 ,,	8·7 ,,
Rheumatism	34 ,,	3·5 ,,
Scarlatina	33 ,,	3·4 ,,
Measles	39 ,,	4· ,,
Enteric fever	11 ,,	1·1 ,,
Tonsillitis	10 ,,	1· ,,
Erysipelas	6 ,,	0·6 ,,
Pleurisy	11 ,,	1·1 ,,
Congestion of lung	9 ,,	0·9 ,,
Delirium tremens	5 ,,	0·5 ,,

more frequent recurrences are noted by other authors (*e.g.* Andral, 16 attacks; Busch, 28 attacks).[1]

In the face of such evidence it is impossible to doubt the generally accepted statement, but we must be content with noting that amongst the cases observed by one of us during ten years' hospital practice, a previous history of pneumonia (*quantum valeat*) was far less frequently obtained than that of bronchitis, or even of rheumatism, amongst the patients coming under treatment for acute pneumonia. In only one instance (out of 123) did the same patient reappear with an attack of pneumonia.

We need not pursue this subject further, but proceed to inquire into the *after-history* of pneumonic patients, as regards the condition of the lungs. It is commonly asserted or implied that the disease is without sequelæ, and certainly in by far the majority of cases resolution is complete before the patient passes from observation, since physical examination no longer reveals any departure from the normal. But we have already seen in dealing with the morbid anatomy of the disease that (rarely it is true) the pneumonic process may eventuate in permanent damage to the lung texture or terminate in suppuration or gangrenous changes. Since the last named can hardly (strictly speaking) be considered parts of the ordinary process, it seems more fitting to consider them as parts of the 'after-history' of the case. In this connection, also, we shall have to dwell at some length upon the relationship which pneumonia bears to pulmonary phthisis.

Before doing so, it may be well to inquire a little into the duration of the process of resolution. The data for determining this are necessarily imperfect, and in many cases, doubtless, the persistence of dulness at the base of the lung is due to pleural thickening, the outcome of the concomitant pleuritis. In the following table we have collected data of the pulmonary conditions ascertainable on physical examination on the last occasion at which such examination was made before the patient's discharge from hospital—the time in most instances dating from the period of crisis. The cases occurred at the Middlesex Hospital during the past ten years (1880–89).

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[1] Other authors might be quoted. 'Like facial erysipelas and acute articular rheumatism, pneumonia is one of those diseases which attack the same person several times, with a certain preference' (Strumpell, 'Text Book of Medicine,' 1887, p. 175). 'An attack affords no protection from subsequent attacks' (Flint, 'Principles and Practice of Medicine,' 6th ed. 1886, p. 161). See also Hilton Fagge, 2nd ed. ii. p. 140.

TABLE SHOWING CONDITION OF LUNGS AS REGARDS PHYSICAL SIGNS AT STATED PERIODS AFTER THE CRISIS IN ACUTE PNEUMONIA.

—	3–7 days	8–15 days	16–21 days	22–28 days	29–35 days	36–42 days	43–49 days	50–56 days
A.—No signs of consolidation, and breath-sounds practically normal; free from râles. 39 cases.	9	15*a*	9a^3	1	1	2a^2	1	1
B.—Râles alone (a few friction?), but no bronchial breathing or dulness. 17 cases.	2	7*a*	2	4*a*	0	1	0	1*a*
C.—Dulness or impaired resonance, with or without bronchial breathing or râles. 36 cases.	7*a*	5	10	5	3	0	2a^2	3a^2 and 1 62dys.

a One case dated from admission.
a^2 Two cases " "
a^3 Three cases " "

Empyema ensued in 4 others. Total, 96 cases.

With respect to the above data, it may be remarked that had it been possible to continue such examination for some few weeks longer, there is little doubt but that the cases comprised under B and C would have come to fall within the category of A. Indeed, there were practically but few cases in group C—viz. those in which the physical signs of consolidation persisted at the close of eight weeks—of which it could be affirmed that the lung would become permanently damaged (chronic pneumonia). Most, if not all, of the others in this category were instances either of delayed resolution or of pleural thickening. From this evidence alone, in spite of its imperfections (for no opportunity has been afforded of an examination of the patient since leaving hospital) it must be conceded that a true chronic pneumonia of any extent is a rare process, much rarer than the chronic interstitial affection secondary to pleurisy, which may be the ultimate cause of bronchiectasis and some cases of non-tubercular 'fibroid phthisis.'[1]

Abscess[2] and *gangrene*[3] of the lung are both very rare as sequelæ; the former being certainly the less common (see Chap. V, p. 88). Not one example of either of these conditions was met with in the series of 123 cases which were admitted into the Middlesex Hospital under the care of one of us during the above-mentioned decade; but, as stated previously, an analysis of 144 *fatal* cases of pneumonia yields 1 instance of abscess and 9 of gangrene. On the other hand it may be of interest to mention that in 38 cases of gangrene

[1] Grisolle had only seen four cases of transition of pneumonia into the chronic state, and also points out how slow some cases are in resolution.

[2] Traube ('Ges. Beiträge.' II, p. 530) records a case of pneumonia, crisis on 15th day, but in whom fever persisted with signs of consolidation. On the 44th day there was expectoration of elastic tissue. The patient also had double parotitis, which subsided without suppuration, and the patient recovered. In another lecture (*ib.* 895) the same writer distinguishes three forms of pulmonary abscess, viz.: (1) very rare, following primary croupous pneumonia; (2) from chronic pneumonia; and (3) cases where there was little local evidence, but marked constitutional disturbance.

[3] Laennec hardly considered gangrene as one of the terminations of pneumonia. Grisolle was of the same opinion, stating that, of 50 cases observed by him in 25 years, in not one was it secondary to pneumonia; and in 70 cases collected by him from writers there were barely 5 which could be so assigned. It seems possible that pneumonic gangrene may be more frequent in other countries, since Morehead (Malabar) and Godineau (Coromandel) have met with it.

collected from post-mortem records 14 were found associated with acute croupous pneumonia.[1]

Except when the lung is perforated and pneumo-thorax set up, it is rarely that abscess is discovered during life, and the differential diagnosis between it and gangrene is often impossible.

The relationship between gangrene of the lung and acute lobar pneumonia has hardly as yet received satisfactory explanation. Laennec considered that the surrounding inflammatory change was purely secondary; and there is, no doubt, much truth in this view as regards many cases. But there yet remain a fair proportion where there seems no room for questioning the fact that acute pneumonia has terminated in gangrene. Of the fourteen cases mentioned as associated with acute pneumonia, not more than eight could fairly be considered as directly dependent on the latter. In the rest, the limitation of the pneumonic areas, the distribution of the gangrenous foci, point to the hepatisation being secondary to the gangrene. At the same time no other adequate cause for the occurrence of the gangrene can be assigned.

That some cases of pneumonia occurring in debilitated subjects, or in those who are the victims of depraved nutrition, may eventuate in gangrene, cannot be denied. Thus in six of these cases there was an admitted history of alcoholic excess, three cases were complicated with granular kidney, and one occurred in a diabetic; but what is the precise determining factor of the gangrenous process in these cases we are unable to say. One can, of course, assume that in all these subjects, as in those where no underlying cachexia is present, there may be such an extension of the inflammatory process as to involve the nutrient vessels of the organ, presumably the bronchial arteries; but we have no actual demonstration of this fact, and we cannot help suspecting that in most, if not in all such cases, there has been admitted into the lung some special irritant or septic material which has initiated the virulent type of gangrenous inflammation. The rarity with which gangrene occurs as a sequel of acute pneumonia is well illustrated by the returns furnished to the Collective Investigation Committee. Out of the 1,065 cases so returned, gangrene resulted only in two, both being old subjects, and one a drunkard.

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[1] For an analysis of these cases see 'Brit. Med. Journ.,' Sept. 5, 1885.

In a certain number of cases, however, the connection between pneumonia and gangrene is more evident. Such are the cases of lobar inflammation, which, instead of resolving, pass into a condition of induration, when the pulmonary texture becomes converted into a dense, more or less vascularised tissue, and portions and tracts of the inflamed lobe may remain so consolidated. Six of the cases from these records are associated with this condition of chronic pneumonia, and amongst them, also, there is the same liability to recurrence in the intemperate and in subjects of chronic Bright's disease. One of the cases appeared to be strictly a syphilitic pneumonia, and in another a fish-bone had been taken into the lung some months previously, and had literally excited a traumatic pneumonia and gangrene.[1] A case precisely similar to this last, where a fragment of chicken bone led to the same results four and a half years after the accident, is related in Dr. Hughes Bennett's 'Clinical Lectures' (p. 689, 3rd edition).

Clinically this sequel of pneumonia is usually recognised by the odour of the breath and sputum, which, from being sickly and offensive, soon acquires the intolerable fœtor of pulmonary gangrene, at the same time physical signs of cavity can often be made out in the unresolved lung and sometimes, owing to rupture of the gangrenous portion, signs of pneumothorax as well.[1]

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[1] Other conditions, such as empyema, when occurring late in the history of pneumonia, may appropriately be included in the after-history of the disease. These we have already considered under Complications, Chapter VI.

The relationship between *phthisis* and pneumonia is not intimate. The processes of tissue degradation and fibroid growth which constitute phthisis do not produce pneumonia, nor are they, except rarely and doubtfully, influenced by that disease. Thus pneumonia, occurring to the subjects of phthisis, will run its ordinary course to its ordinary end without affecting, so far as can be seen, the chronic ailment; while phthisical persons, although they have a morbid liability to ordinary catarrh, are not more exposed to pneumonia than their neighbours.[2]

It may be urged, on the other side, that in a large majority of post-mortem examinations after death from phthisis, pneumonia is noticed along with the caseation or the fibroid induration. Certain parts of the lungs remote from these will be 'red and congested,' or in parts 'hepatised,' or, while one lung is riddled with vomicæ, its fellow will be in 'the first stage of pneumonia.' This is the customary language of such accounts, insomuch that a perusal of them would lead to the conclusion that pneumonia, in some form or other, so far from being an accident, was an invariable concomitant of phthisis. We must note, however, the exact manner of this association.

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[1] A woman, Ellen S., aged 35, known to be of drunken, dissolute habits, was admitted to the Westminster Hospital, April 10, 1890, ten days after a seizure described as pneumonia. The signs above mentioned made the diagnosis both of gangrene and pneumo-thorax sufficiently plain. Post-mortem—in the midst of the consolidation of unresolved pneumonia was a large, ragged, gangrenous cavity opening into the pleura. All the other organs were healthy.

[2] When undoubted pneumonia attacks the subjects of chronic phthisis, the acute disease, both as to its seat and general progress, is quite independent of the other. Both Louis and Walshe state that the mean duration of pneumonia in phthisical subjects is less than when occurring in healthy lungs. 'Some of the most marked examples of rapid resolution I have met with,' says Dr. Walshe, 'were in phthisical persons.' It may be added that the subsequent course of phthisis is no way altered by an intercurrent attack of pneumonia.

In the most chronic forms of phthisis, such even as have been mainly apyrexial throughout, the process of caseation will be found in various stages of progress. There is the excavated vomica, where the work may be said to be finished, and there are ill-defined patches of carnified or solid lung, densest in the centre and shading down insensibly into natural tissue, where it cannot with certainty be said to have begun. These latter are the appearances to which the phrases quoted refer, and they are described as 'the first' or 'the second' stage of pneumonia, according to their degree of density, and in remembrance of what takes place in the acute disease. Yet they represent, not pneumonia, but the several steps in a process which is essentially slow and lingering. During the lifetime of the patient the more recent of these spots have escaped detection altogether. Even to the eye they seldom have the character of pneumonic hepatisation. They are not granular in fracture, nor do they exhibit under the microscope the material of a solid exudation filling air-sacs whose proper structure is unaltered. Sometimes, indeed, this structure is so converted as to bring it within the category of fibroid lung already described.

In acute tuberculosis the lung, in common with other structures, becomes studded with grey, semi-transparent granulations. The true granulation has its seat in the connective tissue, while the neighbouring alveoli, distorted and blocked by the projection of these bodies, are apt, whether as cause or consequence,[1] to exhibit proliferation. A certain number of alveoli may thus be obliterated, but their obliteration is accidental, and not, as in catarrhal inflammation, the main feature of the morbid change.[2]

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[1] Professor Klein, in his work, 'The Anatomy of the Lymphatic System,' Part II., affirms that alveolar catarrh is at the very beginning of the tubercular process.

[2] See Rindfleisch, 'Pathological Histology,' ii. 39.

And, accordingly, acute tuberculosis is seldom recognised at first by any local inflammatory action on the part of the lung. It is judged of rather by general than by special signs. With individuals of a certain disposition or diathesis, nervous depression and high fever, not otherwise accountable, will give rise to a suspicion of tuberculosis. Such cases are often mistaken for typhoid fever, and even when that affection has been excluded the diagnosis rests at last rather upon negative than positive grounds.

And while it is impossible during life to obtain direct evidence of acute tuberculosis, it is to be observed that, in the system of exclusion which finally leads to a correct inference, pneumonia finds a place. We get a step nearer to a conclusion by the observation that the disease is *not* pneumonia. It is a part of the ordinary text-book instruction to point out how, in its physical course, acute pneumonia differs from acute tuberculosis, how in the one the crepitant râle gives place to the sounds indicative of consolidation, while in the other this râle persists, so that days after its first appearance 'the signs of hepatisation are not one iota more obvious than the previous days.'[1] This distinction, which clinical observation witnesses, morbid anatomy fully confirms, yet not without room for error which it may be worth while to notice.

The grey granulation, as has been said, hardly exists apart from catarrhal cell development on the part of the surrounding alveoli. Within this area it is possible to select particular spots where these elements, taken alone, are indistinguishable from those of ordinary pneumonia. Now, along with this catarrh, which is limited to the neighbourhood of the granulations, the entire lung, thus suddenly beset at many points, is apt to become hyperæmic. We have thus, at once, congestion like that of the first stage of pneumonia, and here and there, in isolated spots, sparsely or thickly scattered, the products of inflammation grouped round the several granulations. Yet the likeness to pneumonia, such as it is, is not long maintained. The congestion is sufficiently distinguished by its history. Its origin, duration, and ultimate destiny are all different. The engorgement of pneumonia is but the first step towards the exudation which is the great event of the disease, while this secondary congestion undergoes no such change. The inflammatory products around the several granulations we have already learnt to recognise as the constant accompaniment of local pulmonary irritation.

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[1] Walshe, *loc. cit.* p. 547.

In the next place, it is to be observed of lobar pneumonia that it has no appreciable effect in exciting tubercle. We see children whose family and personal history point most plainly to acute tuberculosis, undergo pneumonia with no further detriment and no more prolonged illness than others. We see them die of it, as others die, the tubercle scattered throughout the inflamed lung not actively concerned in the morbid process.

Regarding pneumonia, then, in its anatomical relations, not as a whole but in bits, not as a disease but as a collection of histological phenomena which may be variously associated, certain of its elements are to be recognised in the proliferative and other changes which accompany caseation and acute tuberculosis. But viewing it in its integrity, in the light of its history, mode of progress, and spontaneous recovery, these same elements occupy a special place and contribute to a special end. Their character is determined by their conduct. The same histological elements in the one place represent the final result of a local disturbance, in the other a single stage in a progressive and orderly series of changes.

There remains still the question whether true pneumonia, unusually protracted, may not at last degenerate into phthisis, forming, so to say, a nidus for the development of tubercle. That pneumonia does so end sometimes, there is strong reason to suspect. Nevertheless, in examining the particulars of cases of phthisis originating, as is asserted, in inflammation, it is quite rare to find a clearly defined, undoubted attack of lobar pneumonia as the starting-point.

Probably with most forms of chronic phthisis the earliest morbid changes concern the alveoli alone. Certainly the fact is that the history of such cases reaches back to a period of ill-health antecedent to any complaint of the nature of acute inflammation. It is not until the phthisical affection is fully declared that sundry of the patient's antecedents are arbitrarily selected to father it. When the patient's history is thus ransacked, undue prominence may be given to certain events of his life, and the account transmitted for our instruction comes through a distorting medium. Yet even so, it is seldom that chronic phthisis can be traced right up to its source in any of those catalogued disorders which are credited beforehand with the power to produce it. The inflammation that is most associated with phthisis is bronchitis, but it is related to it much more as an intercurrent affection than as the initial one, and much oftener in the form of bronchial and pulmonary catarrh than as a true pneumonia.

Again, pneumonia is sometimes set down as the starting-point of phthisis because, in auscultation, the physical signs resemble those of hepatisation. Carried away by that observation, the antecedent history is disregarded. There is a 'latent form' of pneumonia always at hand to do duty on such occasions. Let the patient present the appropriate local signs, and let his illness be recent, and we arrive at once at that diagnosis. The nature of the disease so established it soon obtains, by its persistence, the further name of 'chronic,' and upon its termination in death is supposed to afford a suitable illustration of 'pneumonic phthisis.'

We do not, however, deny that true lobar pneumonia does sometimes end in phthisis. Thus, in rare instances,—usually with persons of phthisical history—the disease will occur in the usual way (or, at least, its physical signs will occur) in one whose health has for some time been impaired. Long after the acute symptoms have disappeared auscultation will indicate that the local products of the disease remain, and that a portion of the lung is still solid. The patient meanwhile either wastes and begins to exhibit the characteristic symptoms of phthisis, or else (and this is less uncommon) he continues for a considerable time, in spite of the local defect, in fair health, the subject of a chronic pneumonia which is not beyond the reach of recovery.

CHAPTER VIII

ELEMENTS OF PROGNOSIS

The mortality of pneumonia in reference to age, sex, and social circumstances—The influence of the seat of lung inflammation, fever, &c.—Pulse indications—Prognosis in respect to complications and the presence of disease in other organs.

At the risk of repetition and of reference to some points yet to be considered, it may be convenient to give a summary account in this place of the elements of prognosis, derived from considerations of age; sex; character and seat of inflammation; associated diseases; mode of origin, and other circumstances, discussed in detail in other parts of this work.

As regards age, when it is said that acute pneumonia is more especially a disease of early life, that its mortality is very small in children, somewhat larger, but still small, in young adults, and increasing with each decade after the age of thirty, it is doubtful whether any addition to this general statement, ample as is the material for it, is of much service in prognosis. That men suffer more often than women, while with the latter the death rate is somewhat the higher, are facts capable of explanation in the differing circumstances of the sexes; they give no warrant for the belief either that there is any extra liability on the part of men, or that, in any given case, the sex of the patient makes any appreciable difference in the chance of recovery.

It is probable that the smallest mortality from lobar pneumonia at any period of life whatever occurs in early childhood. The testimony of trustworthy observers on this point is in practical agreement, and is corroborated by the comparative rarity of the child's hepatised lung in the post-mortem room. But at this early age, as we shall show, broncho-pneumonia is apt to get confused with lobar, and thus the real mortality of the latter gets over-estimated. Add to this that our hospital records comprise only a small proportion of young children, so that, both as to the relative frequency and actual mortality of the disease, we are less well informed in respect to them than to older subjects.

In Huss's Tables (quoted by Dr. Wilson Fox, 'Reynolds' System of Medicine,' vol. iii. p. 689) the numbers below ten years are too small to be worth quoting. Putting these aside, the lowest mortality is between twenty and thirty years, 1,041 cases giving 61 deaths, or something under 6 per cent. From ten to twenty, with a much smaller number of patients, namely, 226, the death rate is a little over 6 per cent. From thirty to forty years of age shows a mortality double that of the preceding decade, 816 cases furnishing 97 deaths, or nearly 12 per cent. From forty to fifty the mortality is nearly 20 per cent., and from fifty to sixty it is over 21 per cent. Above sixty the cases are too few to be of service.

With these results the 'Collective Investigation Committee's Report' is in general agreement. Of 1,039 cases tabulated (687 men and 352 women, nearly two to one) the largest aggregate number of patients falls between five years old and thirty, and the largest number for any period of five years is sixteen to twenty, namely 117. The mortality of this same period, moreover, is the lowest of all, that is 5·1 per cent. The ratio of mortality between twenty and thirty more than doubles this percentage, 215 cases (143 men and 72 women) showing a death rate above 12 per cent. Mortality again rises, notably over thirty-five years of age and from fifty-one to fifty-five shows the largest percentage of all, old age excepted, namely 39·5.

As in other statistics, so here, men are attacked more often than women, the disparity being most marked between forty and fifty. There is also a somewhat higher rate of mortality on the female side, namely, 19·9 per cent. as against 14·2 males, a proportion nearly agreeing with that of Huss.[1]

SEX, AGE, AND MORTALITY.—'Coll. Invest. Record,' vol. ii. p. 29.

Years	Males			Females			Totals		Total	Mortality per cent.
	Rec.	Died	Total	Rec.	Died	Total	Rec.	Died		
— 5	34	3	37	23	4	27	57	7	64	10·9
6–10	64	6	70	32	—	32	96	6	102	5·9
11–15	58	2	60	35	6	41	93	8	101	7·9
16–20	80	4	84	31	2	33	111	6	117	5·1
21–25	72	7	79	30	5	35	102	12	114	10·5
26–30	56	8	64	30	7	37	86	15	101	14·8
31–35	46	10	56	24	5	29	70	15	85	17·6
36–40	41	13	54	20	7	27	61	20	81	24·7
41–45	35	17	52	5	3	8	40	20	60	33·3
46–50	24	14	38	19	4	23	43	18	61	29·5
51–55	17	9	26	8	7	15	25	16	41	39·5
56–60	14	7	21	7	2	9	21	9	30	30·
61–65	11	7	18	8	2	10	19	9	28	32·1
66–70	13	4	17	7	7	14	20	11	31	35·5
71–75	1	6	7	4	4	8	5	10	15	60·8
76–80	—	—	—	—	3	3	—	3	3	
81–85	2	—	2	—	1	1	2	1	3	
86–90	2	—	2	—	—	—	2	—	2	
Age not stated	14	3	17	2	2	4	16	5	21	—
Totals .	584	120	704	285	71	356	869	191	1,060	—

With these tables, by way of contrast, it may be useful to cite some figures derived from the practice of a general hospital. There is a certain advantage in this, owing to the differences in social status and surroundings which such patients present from those whose histories were collected by the Investigation Committee. At the same time it must be borne in mind that a hospital naturally attracts severe cases, drawn from the worst surroundings, and that a certain proportion are admitted in a moribund state. We have selected for this purpose the statistics of the Middlesex Hospital for a period of twenty years (1869 to 1888), as given in the published annual Reports of the Medical Registrar. The total number of cases of acute pneumonia admitted into the hospital during this period was 1,010, or about 50 less than those gathered by the Collective Investigation Committee, and it is remarkable that the gross mortality approaches to within 1 per cent. of that given by the Committee. If it be permissible, in the presence of such large numbers, to draw any inference from these figures, they would tend to show that, as regards the mortality rate, external surroundings have comparatively little influence in this disease. Nevertheless, when we inquire more narrowly, and contrast the mortality in the two sexes, we find that the rate among males is much higher in the hospital cases, whilst among females the proportions are exactly reversed. An explanation for this divergence may perhaps be found in the fact that the cases of illness in the male sex sent to hospital are more serious than those of females. The figures are as under:—

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[1] In Dr. Longstaff's Memorandum on the Incidence of Fatal Pneumonia, based on more than 24,000 cases ('C. I. C. Rep.' p. 102) males suffer more than females, as 3 to 2, the difference being most marked between thirty-five and sixty-five, where it is as 2 to 1.

—	Rec.	Died	Total	Mortality per cent.
Males				
Collective Investigation Report . .	584	120	704	17
Middlesex Hospital Statistics . .	520	147	667	22
Females				
Collective Investigation Report . .	285	71	356	20
Middlesex Hospital Statistics . .	298	45	343	13

Collating the two series, we obtain a mortality rate in the two sexes :—

Males . . 1,371 cases, 267 deaths ; or 19·4 per cent.
Females . . 699 ,, 116 ,, ,, 16·7 ,,

which may be fairly taken as representing the average mortality, and therefore the prognostic significance of an attack of pneumonia in the male and female sex respectively.

A like comparison may be made with regard to the influence of *age*, upon which we have dwelt above in connection with the Collective Investigation figures alone. Here again, by combining the two series, we may hope to arrive at a juster conclusion, which, after all, can only be attained by an appeal to statistics. It will be seen that the death-rate remains at about 5 per cent. until after the age of twenty, and that it then rises, almost in arithmetical progression, during the next two decades,[1] reaching, between forty and fifty years, the enormous rate of 39 per cent., which, except for the very aged, is the highest rate of mortality recorded. These facts are sufficiently illustrated in the accompanying table and diagram :—

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[1] During five years (1884–1888) 247 cases of pneumonia were admitted into the *Westminster Hospital*, namely, 180 males and 67 females ; 52 died, about 20·9 per cent. The relatively large mortality is accounted for by the fact that the largest number for any decade (namely, 62, or a quarter of the whole) fall between 30 and 40, the period at which, according to all statistics, the death-rate of pneumonia undergoes considerable increase.

In the preparation of the 'Report on Pneumonia' for the Collective Investigation Committee we obtained the statistics of mortality from most of the London hospitals, but in all but the following the numbers given were too small for any estimate of percentage :

St. Bartholomew's, 137, with 28 fatal ; 20·4 per cent.
The *London Hospital*, 100, with 20 fatal ; 20 per cent.
St. George's Hospital, 86, with 18 deaths ; at the rate of 20·9 per cent.
At *Guy's Hospital* only 10 patients died out of 62.

It is not to be supposed, however, that the death-rate is as uniform as the above figures indicate. Thus, the mortality of pneumonia at the *London Hospital* in 1875 was a little under 24 per cent. ; in the year following it was nearly 39 per cent. ('Coll. Investig. Record,' vol. ii. p. 51).

It would be obviously unfair to infer that hospital mortality is an exact reflection of the death-rate of pneumonia generally. The destitute and the drunken (the worst subjects for the disease) form a larger proportion of hospital than of private patients, while a certain number admitted in a dying state have been wholly neglected in the earlier stages of their illness.

The death-rate when limited to early life will be considered in a chapter devoted to children.

MORTALITY TABLE AT DIFFERENT AGES. BOTH SEXES.

Years	Recoveries			Deaths			Total	Mortality per cent.
	C. I. Rep.	Mid. Hosp.	Total	C. I. Rep.	Mid. Hosp.	Total		
—10	153	183	336	13	5	18	354	5·
—20	204	228	432	14	11	25	457	5·4
—30	188	191	379	27	39	66	445	12·6
—40	131	101	232	35	46	81	313	25·8
—50	83	56	139	38	51	89	228	39·
—60	46	37	83	25	25	50	133	37·6
—70	39	14	53	20	9	29	82	35·3
Above 70	9	2	11	14	1	15	26	57·7
Age not stated	16	6	22	5	5	10	32	—
Totals . .	869	818	1,687	191	192	383	2,070	—

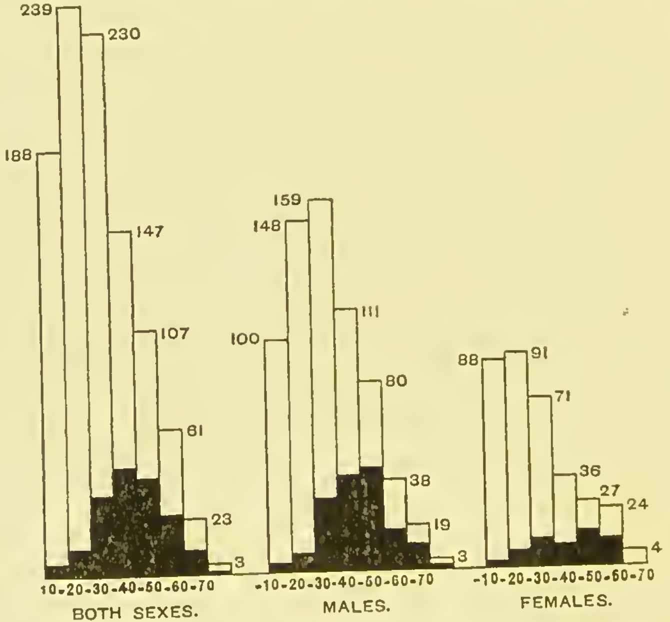

FIG. 9.—Diagram of the mortality from acute Pneumonia, at different decades of life in the two sexes, based on 999 cases admitted into the Middlesex Hospital, 1869–1888. N.B.—The total number of cases was 1010, and of deaths 192; but in 11 the ages were not recorded.

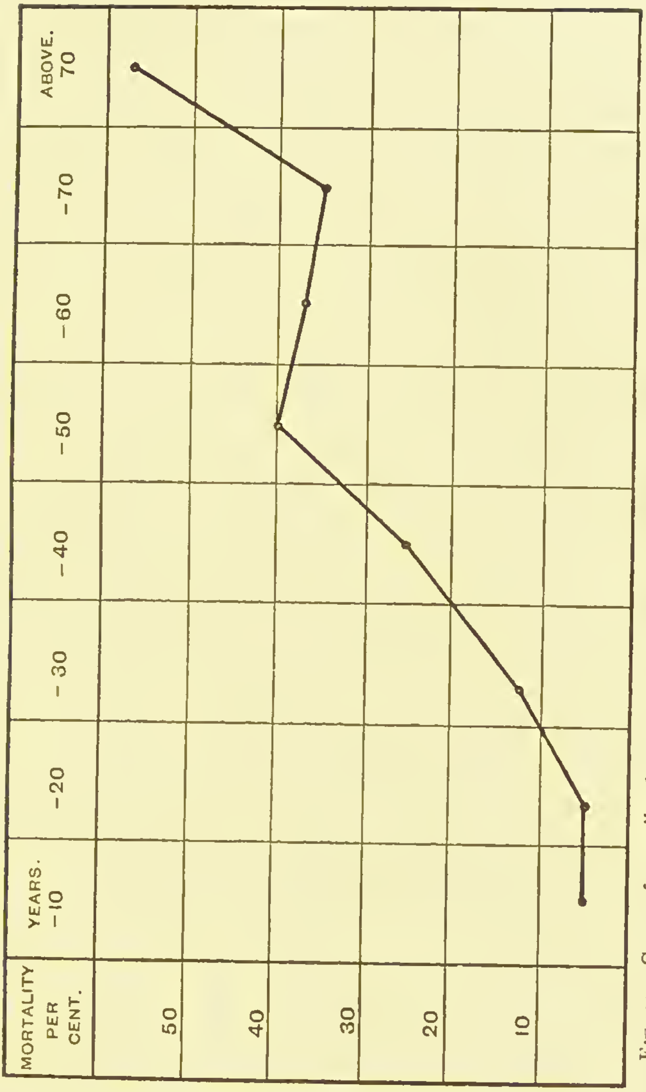

FIG. 10.—Curve of mortality from acute Pneumonia at different age periods (based on analysis of 2,038 cases).

Before leaving the question of mortality, which we have been discussing at tedious length, allusion must be made to recent attempts (notably by Dr. H. Hartshorne, in a paper read before the College of Physicians, Philadelphia, February, 1888), to demonstrate that the mortality from pneumonia has not only not diminished with the progress of knowledge, but that on the contrary it has risen considerably since the early decades of the century. On this belief it has even been maintained that the abandonment of the old heroic and active plan of treating the disease is responsible for an increased lethality. We need hardly say that we do not share this view, which will, however, be more appropriately discussed in a later chapter. It may suffice now to point out that the statement is based on fallacies, of which an admirable exposition has been given by Drs. Coolidge and Townsend in a recent paper,[1] embracing a careful analysis of the records of the Massachusetts General Hospital from 1822 to 1889. The authors found that, although the recorded mortality from pneumonia had risen from 10 per cent. in the first decade of this period to 28 per cent. in the present decade, yet that this could be readily explained, not by any 'change of type' or varied treatment of the disease, but by the simple fact that with the progress of years a greater proportion of the aged, delicate, and intemperate have been admitted into the hospital. Excluding such subjects, the decennial mortality rate would have been almost stationary throughout this long period. It may be remarked that they deal with 1,000 cases: a number closely approximating that above given from the twenty years records of the Middlesex Hospital.

One further remark is necessary. It will be shown in a later part of this work that the small mortality of pneumonia in early life is in some degree accidental. Age must be considered in conjunction with habit. The modes of life which most prejudice recovery—especially alcoholic intemperance—are not contracted in childhood, nor, with rare exceptions, in youth. That the disease is, on the whole, more fatal between 30 and 40 than in the preceding decade is a circumstance that, taken alone, need hardly influence prognosis; it is partly explained by the larger number of intemperate people comprehended in the later age. Again, broncho-pneumonia, hypostatic consolidation, and other conditions to which we shall presently refer, cannot, with certainty, be excluded from statistical tables, and they tend to misrepresent both the frequency and the death-rate of true lobar pneumonia, especially with infants and elderly people.

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[1] 'The Mortality of Acute Lobar Pneumonia,' a paper read before the American Climatological Association, June 1889, and published *in extenso* in the 'Medical News,' July 27, 1889.

In estimating the probabilities of recovery or death in any given case, there are obviously many considerations of greater weight than those derived merely from a study of mortality statistics. The latter can only deal with generalities, the former embrace the numerous and varied individual circumstances which, after all, form the main grounds of prognosis.

We have, then, to inquire what are the unfavourable indications as regards—1st, the attack itself; 2nd, the part of lung involved; 3rd, the degree of fever; 4th, the existence of complications and of disease in other organs which endanger recovery. Of equal moment are—5th, the habits and other circumstances of life special to the patient at the time of his attack. In many of these respects prognosis is governed by much the same factors as in the case of any other acute febrile disease.

1. At the outset there is little to indicate the probable course to be taken by the affection; for, although the illness may set in with great severity and the fever rapidly reach a considerable height, yet it is by no means certain that the subsequent course of the disease will be as severe. No one would venture, then, any confident opinion at this early stage. It is only after a few days have elapsed that indications appear which are of real prognostic value. Often, indeed, the period of the 'crisis' is in all senses the critical period. Thus, it was shown in the

Collective Investigation returns that the fatal day mostly occurred from the 4th to the 11th (in 133 out of 175), the 6th, 7th, 8th, and 10th being the days on which death most commonly took place, or the very periods at which, in cases of recovery, the crisis most frequently happens. Consequently, it is not surprising to find that in the majority of the fatal cases death occurs during a more or less marked decline in the fever.

2. It is a point of some interest, but also of some difficulty, to determine the prognostic significance of involvement of right or left lung, of particular portions, or of the two together. Thus it has been held that pneumonia of the left lung is more serious than that of the right; and that apical inflammation is graver than basic. In this, as in all such questions, the difficulty lies in instituting a just comparison between cases. So many individual circumstances, not only as to age and sex, but as to constitutional condition, interfere in the formation of sound opinion, and serve to explain the discrepancies of statements on the point. Statistics are thus of but partial use, and at most should only be employed with reserve. Our own experience suffices to show that, while no distinction whatever can be made as between the left side and the right, apex pneumonia is not so fatal as basic; and, further, that it does not, as is often supposed, subject the patient to the risk of tubercular phthisis.

That there is, *cæteris paribus*, an increased gravity in the case of involvement of the greater part of one lung, or of double pneumonia, as compared with cases where the inflammation is limited to a small region or to one side, no one can venture to deny. The facts of the post-mortem room demonstrate this, and, indeed, it is almost self-evident. The statement by the Collective Investigation Committee is, we believe, in general accordance with the experience of all physicians. 'The rate of mortality where both lungs are involved is . . . more than double that where one side only is affected, the proportion being 7 to 3·3. There is no appreciable difference in respect of mortality between the two sides. . . . Mortality of right base is 1 in 8; of left, nearly 1 in 9. The highest rate of mortality with reference to site next to that attributable to both sides belongs to both bases; the next highest to the whole of the right lung.

Affection of 'the whole of one lung,' hardly to be taken literally, is fatal in 1 in 4·3 instances (upon the showing of 111 cases), and affection of one apex fatal in 1 in 7·3 instances (upon the showing of 89 cases).' Huss found the mortality of bilateral pneumonia to be 1 in 4; that of left-sided pneumonia 1 in 11; and of right-sided, 1 in 13; whilst Grisolle, by a careful selection of cases, otherwise comparable, could find no difference in mortality amongst those in whom the right or left lung was involved, whilst he met with 7 deaths in 16 cases of bilateral pneumonia, these latter, however, as he pointed out, were in older subjects than the unilateral cases. On the other hand, all these statements need to be modified by the admitted fact that extent of local inflammation is not more important as a factor in prognosis than is the degree of general constitutional disturbance. The evidences of such derangement are practically of more value in estimating prognosis than the signs of the more or less wide extent of lung implication. There are many fatal cases where the hepatisation is limited to one lobe, and, on the other hand, many cases of recovery where it has been very extensive.

3. Indications far more to be relied on are those derived from the *temperature* and *pulse*, especially the latter. The continued high fever of pneumonia is in itself an element of danger, and the fatal issue, which, as we have seen, commonly takes place just at the time when otherwise the fever would have run its course, is chiefly due to the inability of the organism to withstand longer the effects of this pyrexia. Hyperpyrexia is very unusual, but ordinarily the range of tem-

perature is high, and the longer the crisis is delayed the greater the gravity of the outlook. Again, as we have shown, the perturbation of the crisis itself may suffice to overcome the resistance of the organism, enfeebled as it is by the preceding fever. In such cases death is due to asthenia, or exhaustion; it is rarely due primarily to asphyxia from the lung affection. This being so, we find in the character of the *pulse* the chief indications of the probable termination of the case, and we cannot too strongly urge the importance of its careful observation. An increasing rate and smallness of the pulse mean that the heart, already embarrassed in its action by the disordered pulmonary circulation, is further weakened by the pyrexial state. A pulse rate above 120 is serious, one of 140 to 150 of almost fatal import; whilst the weakened, fluttering, or imperceptible impulse of the heart, and its enfeebled, shortened first sound, afford additional proof of the degree to which this organ is affected. Irregularity or intermittence of the pulse are also very grave signs. It is from its effect on the heart that the complication of acute pericarditis is so dangerous.

Among other obviously unfavourable signs may be mentioned prostration, prolonged delirium and insomnia, tremors, a dry, brown tongue, coldness of extremities, cyanosis, with indications of progressively increasing engorgement of the non-hepatised parts of the lung. Such engorgement is in the main referable to the failing cardiac power, so that the general conclusion is again emphasised—namely, that it is to the heart that we must chiefly look for indications of the probable outcome of the case.

4. The issue of pneumonia is very largely affected by the cò-existence of *disease in other organs*, either as complications of the pneumonic attack or as conditions which have been antecedent to it, and which in a sense may be held to have

predisposed to it. The remark of Dr. H. G. Sutton[1] embodies much truth—namely, that *fatal* pneumonia occurs mostly in individuals who are already the subjects of some tissue degeneration or debility, and that in comparatively few cases of death from pneumonia is there lack of evidence (post-mortem) of antecedent disease of the lung. The extent to which such complications modify the course of pneumonia has been shown by Huss in a statistical form, his researches tending to point to the graver influence of *emphysema* than of *bronchitis*, either acute or chronic, although in the presence of each of these conditions the mortality is above the average.

The existence of *phthisis*, as we have said, is not necessarily of bad omen. It depends entirely upon the stage of that disease at which the patient has arrived when attacked by an intercurrent lobar inflammation. Amongst the rarities of pneumonia is the outbreak of *acute tuberculosis* simultaneously with its onset, of which we have recently seen an instance. Of course such a complication is absolutely fatal. The prognosis in cases complicated by pleural effusion and by *empyema* is not unfavourable so far as our experience goes, although Coolidge and Townsend (*loc. cit.*) give three fatalities out of six cases so complicated. It is otherwise with cardiac complications. *Acute pericarditis*, as we have shown, is very serious; it is met with in a fair proportion of the fatal cases, and its presence may escape detection during life. Of *endocarditis* it is less possible to speak with certainty, but the liability to the malignant or ulcerative form must not be forgotten. Again, chronic valvular disease materially enhances the danger of the attack. Nevertheless, there are many subjects of cardiac disease who pass unscathed through an attack of pneumonia. It is not the mere presence of valvular obstruction or incompetence which would influence the prognosis, but rather the indications of the degree to which the valvular defect had been compensated by hypertrophy and dilatation. It is now well established that a valvular defect may be borne for years without any subjective symptom of heart disease, and although the supervention of an acute illness may suffice to break down the compensation, and especially an illness which throws such additional strain on the heart as does pneumonia, yet even under these circumstances no appreciable injury may be inflicted on the already damaged organ. There are instances, known to all, where the signs of mitral obstruction or regurgitation have existed prior to a pneumonia which has yet run its course without producing any notable increase of cardiac symptoms. At the same time, it is hardly possible to say that some deterioration has not been initiated, which will show itself at some subsequent time. This, however, bears more upon the prognosis of heart disease than of pneumonia; and the most that can be said on the latter head is, that cardiac affections do, to a certain extent, modify the course of pneumonia unfavourably, and that in some cases without doubt the life of the subject of cardiac disease is cut short by reason of the failure of the organ to bear the double strain.

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[1] 'Medical Pathology,' p. 82.

Of all diseases complicated by pneumonia none have so serious an effect as *chronic Bright's disease.* Pneumonia in the subjects of either form of this affection is, as we have already shown, of great gravity. In further proof of this, it is only necessary to glance through the post-mortem records of a series of cases of pneumonia, and to note the comparative frequency with which 'granular' kidneys are mentioned. At the same time it must be admitted that the subjects of renal disease, speaking generally, do not die from pneumonia. The two statements may seem contradictory. They are not so in reality. Chronic Bright's disease is a wide phrase. It is met with in the post-mortem room twice as often as it is admitted into the hospital ward. We think it will be found by any impartial investigator that the patients dying of pneumonia with granular kidney are just those in whom the renal disease has not been prominent during life. At least that would seem to follow from the fact that 'granular' kidney occurred in 29 cases of fatal pneumonia out of a total of 144 autopsies in that disease, or 20 per cent., whilst of 77 examinations of declared chronic Bright's disease there were only 5 in which lobar hepatisation was met with, or about 7 per cent. The latter observation by no means conflicts with the former. Rather it emphasises the point in question, that the existence of renal disease is a real danger to the pneumonic patient.

The same is hardly true of *acute nephritis*, an occasional complication of pneumonia, which, as we shall see, is not seldom recovered from (Chap. XI). Nor is it true of the prognostic significance of albuminuria to which allusion has been made in an earlier chapter (*vide* p. 35).

Delirium tremens is a formidable complication. Huss found that about 1 in 14 affected with it died. Coolidge and Townsend give 15 deaths among 23 cases—an enormous proportion. We should incline rather to this latter mortality as being nearer to the truth. The experience of our Metropolitan hospitals goes far to show that the conjunction of pneumonia and delirium tremens is most ominous. But to attribute all the fatality to the pneumonia is not accurate. It must be borne in mind that the patients suffering from delirium tremens are those whose tissues and organs are all more or less damaged by the effects of alcoholism; they are the subjects, that is, of visceral disease, and on that account the least able to withstand the fever and its effects.

Here it may be convenient to allude to the question of *alcoholic intemperance* as influencing prognosis. There can be no doubt that such habits not only predispose to pneumonia, as we shall show presently (Chap. XVI), they also increase its severity. To use a current phraseology, alcoholism favours the morbidity as well as increases the mortality of this disease. It is, however, not easy to bring the matter to the crucial test of statistics. This was attempted in the Collective Investigation returns, but with only partial success. It came out that whereas the 'total abstainers' had a mortality of 10·4 per cent. (267 cases), and the 'temperate' one of 17·4 per cent. (655 cases), the 'intemperate' gave a mortality of 42·8 per cent. (105 cases). Obviously, however, such statistics are vitiated by the inclusion of children under the first head, as well as by the elasticity of the terms employed to denote drinking habits. Drs. Coolidge and Townsend give a mortality of 15 per cent. among the 'temperate' (only 55 cases), and of 41 per cent. among the 'intemperate' (109 cases). In any case a history of excessive indulgence in alcoholic drink materially adds to the gravity of the prognosis.

Of other complications there is little that need be said in respect to their prognostic significance. *Meningitis*, which rarely occurs, is invariably fatal. *Cirrhosis of the liver* is frequently met with in cases where intemperance is a factor, and doubtless its presence increases the tendency to death. On the other hand, the symptom of jaundice is not to be considered as of itself of bad augury. Unless associated with cirrhosis it is mostly transient and of slight significance. Of the *acute specific diseases*, typhus is the most to be dreaded. Typhoid is rarely so complicated, and no facts sufficient to draw conclusions are available (Chap. XI). Erysipelas is occasionally complicated with pneumonia or may arise in its course, but we agree with Huss that it does not greatly add to its gravity.

5. It would be an exaggeration to say that pneumonia always spares the previously robust, or, in other words, that

in fatal cases it is never uncomplicated. But it is certain that if we could separate all the examples of the disease in which there was no evidence of any other lesion than those attributable to the acute process itself, the amount of fatality in the latter category would be small. Huss places it at only 5·79 per cent., as contrasted with a rate of 19·29 per cent. among the complicated. It is true that the disease is more fatal to the weakly than to the strong, but it is to be remembered that there are cases where young and vigorous subjects, who have never ailed before, and in whom there is no inherited tendency to pulmonary disease, have been carried off by pneumonia. In such subjects we are prone to think that the disease runs a more 'sthenic' course, but this by no means implies that it will be a favourable one. They have the advantage over those who are already debilitated, yet in these latter the attack may have a comparatively mild course, and the 'wind be tempered to the shorn lamb.' For all this, it is a truism that the chances of recovery are the less for one who is weakly than for one who is robust. The vital resistance to acute disease is lowered by many circumstances. Anxiety and trouble are often as marked factors in its production as is physical weakness. We shall endeavour to show, when speaking of the etiology of the disease, that muscular fatigue, exposure, antecedent depressing illness (of which we have an example in influenza) will not only predispose to an attack of pneumonia, but add to its gravity. All these considerations, together with others which concern the special surroundings of the patient, must be taken into account in attempting to forecast the probable course and termination of his illness.

Lastly, there is evidence to show that in *particular seasons and places* the type of the disease may be more fatal than at others. The varying rates of mortality from year to year seem to demonstrate this. We know, too, of its occurrence occasionally in epidemic form (Chapter XIII), and that some of these epidemics have been marked by a fatality far greater than has been the case with others. Take, again, the instances (though rare) where contagion has been credited with the spread of the disease in a household. The tale of mortality which some such histories report is very striking. We are thus forced to the belief that, as with the specific fevers so with pneumonia, there is a variable tendency to a fatal issue owing to conditions which are as yet, for the most part, matters of conjecture rather than of proven fact. A generation back the medical world was occupied with the theory of the 'change of type' of diseases, the race was becoming more enfeebled, and disease shared in the weakness. We can dismiss such theories with confidence, but still we are confronted with facts which require theories as indefinite for their explanation. And one of these facts is the undoubted variability in the severity of acute pneumonia at different times, the recognition of which must influence prognosis in every case.

CHAPTER IX

ILLUSTRATIVE CASES

THE following abstracts of cases derived from our own practice are intended to exhibit certain features of simple pneumonia to which reference has been made in the preceding pages. They illustrate the ordinary course of chill pneumonia arising suddenly in a healthy person ; the nervous symptoms that sometimes attend the disease, especially when the upper lobe is involved ; the occasional delay of the physical signs ; the uncertainty of prognosis anterior to crisis ; the occasional persistence of pneumonic consolidation ; the fallacy of temperature taken alone as a means of prognosis ; departures from the usual type of pyrexia ; the rapid fatality of some cases without obvious cause and contrary to the common rule ; the recovery of others by crisis notwithstanding the gravest symptoms ; and, lastly, the occurrence of certain complications in the course of the disease.

Such cases as may be necessary hereafter to illustrate other varieties and associations of pneumonia will be incorporated in the text and can easily be found by reference to the index.

CASES.

I. Chill pneumonia ending by crisis on the sixth day.

II. High temperature ; venesection ; crisis delayed till fifteenth day.

III. A case of great severity, but with early crisis.

IV. Apex pneumonia with active delirium.

V. The same with delayed physical signs, extreme prostration and late crisis.

VI. The same with melancholia.

VII. Double pneumonia with persistence of physical signs.

VIII. Intermittent temperature in pneumonia.

IX. Pneumonia with hæmoptysis ; slow pulse, intercurrent bronchitis.

X. Pneumonia fatal within ten days ; highest temperature, 103·2.

XI. Pneumonia following accident, fatal six days later and within three days of physical signs.

XII. Pneumonia spreading from its first seat. Symptoms resembling crisis. Death on seventh day.

XIII. Pneumonia followed by empyema ; drainage ; recovery.

XIV. Pneumonia followed by empyema ; drainage ; recovery.

XV. Apex pneumonia ; pleurisy with effusion ; suppuration of parotid gland.

XVI. Pneumonia ; cerebral meningitis ; death.

CASE I

Chill Pneumonia, ending by crisis on the sixth day.

Stephen B., aged 22, admitted into the Westminster Hospital February 21, 1871. Three days before, while crossing Westminster Bridge (the day being cold and gusty), and in perfect health, he was suddenly seized with 'stitch' ; he at once took to his bed, and was carried from it to the hospital.

On admission he has the dusky florid look and pained expression of pneumonia ; the sputa are rust-coloured, but as yet very scanty ; a little unmixed blood is also spat up ; pulse is 108, temp. 102·2. The lower part of the left chest is dull to percussion, and yields increased vocal fremitus; breathing in this situation is purely tubular. At the upper part of the same side, above the dulness, there is crepitant rhonchus.

On the 23rd (or sixth day of his illness), the pulse had risen to 120, temperature was 102·1. There was no urgent symptom. The man was now sweating profusely.

On the 24th (the fourth day from admission, and seventh of illness), the pulse was 93, or 27 below the previous day, and temperature 101·8.

On the 25th redux crepitation had nearly replaced the tubular respiration; the man was as good as convalescent; the pulse 72, temp. 99·2.

The pulse, continuing to fall, became at last remarkably slow; on March 1, eleven days from the first seizure, it was 48, when the patient was practically well, although some dry cough lingered.

The above sketch illustrates in outline some of the leading features of ordinary pneumonia, as described in Chapter III. Its apparent origin in exposure, its sudden and severe access, its crisis, equally sudden, about the sixth day, followed by sweating. The fall of the pulse in this instance was much more marked than that of the thermometer. And (as occurs sometimes in enteric fever also) the pulse-rate succeeding the pyrexia was abnormally slow. The case did not call for stimulation, and did perfectly well left to itself.

Case II

Pneumonia (left base) – High temperature—Intercurrent pleurisy of opposite side--Venesection—Crisis on fifteenth day.

Julian L., aged 15, an errand boy, was admitted into the Middlesex Hospital on July 6, 1882. A well-nourished, muscular lad, he had from childhood been subject to bronchitis, for which he had frequently been treated in the hospital, the last occasion being two years ago. His present illness commenced on July 2 with several attacks of shivering, a sharp cutting pain in the left side of chest, headache and giddiness. He attempted to work next day, but becoming worse was obliged to abandon it.

On admission (*fifth day of disease*) he complained of a stitch in the left side, and had a frequent dry cough. The breathing was short, jerking, 48 per minute. The chest was everywhere resonant on percussion, but at the left base the breath sound was harsh, almost bronchial, and accompanied by fine crepitant râles. Vocal resonance comparatively increased at this base. The heart's apex-beat was in the normal position, and a soft systolic bruit was audible at both apex and base; the second sound clear and sharp. Pulse 120, full, bounding. Skin dry; temperature 102·8. Tongue thickly coated, moist; bowels confined.

The temperature was at first taken hourly, and at 7 P.M. it was 104°, falling to 103·6 after the body was sponged with cold water; but at 10 P.M. it rose to 105° and remained at a high level during the night, which was passed restlessly, with delirium towards the morning. Under the influence of sponging the temperature fell to 101·6 at 10 A.M. on the 7th.

July 7 (*sixth day*).—Still severe pain in left side; aspect depressed; face flushed. Bowels have been freely open. There is now marked dulness over lower two-thirds of left back, the breath sound being bronchial and accompanied by fine crepitation. In front, below clavicle, the resonance is skodaic. No expectoration. Throughout the day the pyrexia remained high—reaching 105·2 at 6 P.M., when sponging again practised. Pulse 120. Resp. 44.

July 8 (*seventh day*).—He passed a quiet night. The cough is now accompanied by expectoration—rusty in character. The dulness over left back has extended as high as the spine of scapula. Temperature much lower (100·2 to 102·6). The pulse-frequency has also fallen considerably, viz. to 96; and respiration to 36.

July 9 (*eighth day*).—Cough frequent; sputa rusty. No change in physical signs; Skodaic resonance well marked over upper left front and axilla. A crop of labial herpes has appeared. Temperature between 102° and 103·8. Pulse 96. Resp. 60.

July 10 (*ninth day*).—Still high fever, the temperature twice reading 104·8; very flushed, *alæ nasi* working. Very restless during night. Pain in side severe, and leeches applied with relief. Urine, sp. gr. 1030, acid, no albumen. Pulse 96. Resp. 48.

July 11 (*tenth day*).—Pain now on right side, where, at base, fine friction sounds are audible. He had a restless night, and has considerable dyspnœa (resp. 54). Pulse small, 120. Face very flushed; lips dry; tongue thickly coated. Distinct recession of lower interspaces with inspiration on both sides. Some impaired resonance at right base. Friction at left base as well as right.

In the afternoon he became very cyanosed after a fit of coughing. It was decided to perform venesection. This was done by

Mr. Lyell, 10 ounces of blood being withdrawn from the right arm. Very little effect on the pulse. Fever high, the temperature at 1 P.M. again reaching 105°, and not falling much till the following morning.

July 12 (*eleventh day*).—Very restless during night; cough troublesome, paroxysmal; and after coughing he becomes very cyanosed. Dyspnœa more marked. (Resp. 60.) Pleuritic friction still marked at right base. Very little alteration in physical signs over left lung. Pulse 120. Temperature was for most part above 103; maximum 104·2.

July 13 (*twelfth day*).—The bowels are open twice daily. Breathing less laboured, and cyanosis not so marked, except after a fit of coughing. This is still very troublesome, but sputa are no longer rusty. Friction more marked at right base. Signs of consolidation of left lower lobe persist. Temp. 101·4 to 104·2. Pulse 120. Resp. 48.

July 14 (*thirteenth day*).—Cough less; sputa muco-purulent. Dulness over left back now diminishing, and breath sound no longer tubular; crepitation abundant. Friction still audible at right base. Pulse 120. Resp. 54.

Although the pulmonary inflammation was thus apparently subsiding, there was no abatement of pyrexia; at 5 P.M. the temperature was 105°, at 7 P.M. 105·6, and this in spite of the administration of tinct. aconiti in drop doses every quarter-hour for four doses, and then every half-hour for eight doses. With the access of high fever he began to suffer from diarrhœa.

July 15 (*fourteenth day*).—There was a continued and decided fall in temperature during early morning until 9 A.M., when the minimum of 100° was reached; then it rose again to a maximum of 104·2 at 9 P.M. Sweating. Bronchitic signs in chest. Diarrhœa checked by starch and opium enema.

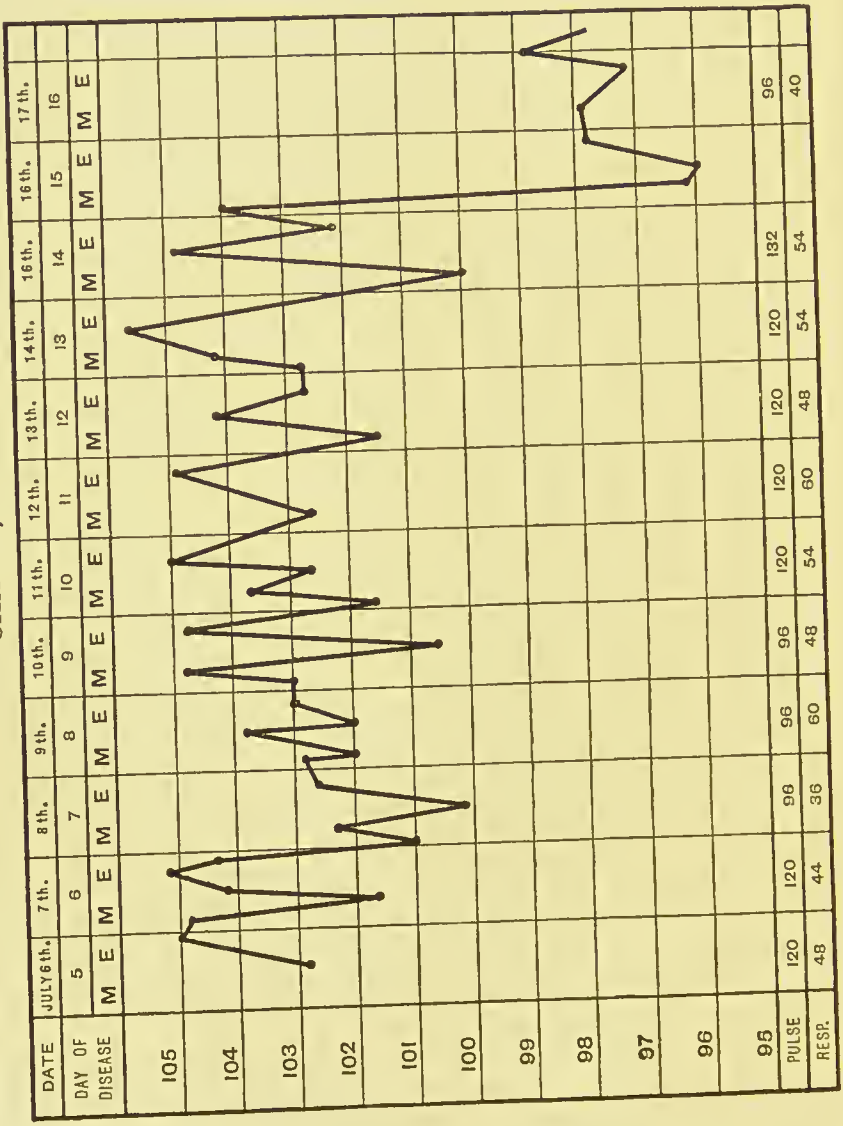
July 16 (*fifteenth day*).—On this day a definite crisis occurred, the temperature subsiding from 104° at 1 A.M. to 101° at 5 A.M., and 96·2 at 11 A.M., and 96° at 1 P.M. (or a fall of 8° in twelve hours). It remained subnormal throughout the rest of day and night. With the onset of crisis he had a severe attack of vomiting, and afterwards slept much (Chart 7).

July 17 (*sixteenth day*).—He passed a very quiet night. The cough is still troublesome; sputa muco-purulent; sonero-ribilant rhonchi and moist râles audible in all parts of chest; signs of consolidation at left base have passed away. Still some diarrhœa. Temp. 97° to 98°. Pulse 96. Resp. 40.

July 18 (*seventeenth day*).—Sleeps well, and able to take more food. Temp. 98° to 98·4. Pulse 72. Resp. 30. But bronchitic signs increased, and in the evening had an increase of dyspnœa.

From this date he continued to improve. By the 22nd he was

CHART 7.



practically free from bronchitis. On the 27th he was allowed to get up, and on the 30th to go into the garden. He left the hospital on August 10.

The interest of this case centres around the long continuance of pyrexia, which was of marked severity. The type of the disease was essentially sthenic, and the apparent extent of lung involvement was hardly commensurate with the high fever. At the same time it is possible that, underlying the pleurisy of the right base, there was some pneumonic change, although this was not revealed by physical signs. This might explain the delayed crisis, and also the fact that, when it occurred, resolution had already commenced in the left lung. Recourse was had to venesection in consequence of increasing cyanosis, but its performance was not followed by any appreciable relief to the dyspnœa, nor had it the least effect upon the pyrexia.

CASE III

Pneumonia of right upper lobe—Rapid development—High, variable temperature—Pulse-respiration ratio—Crisis on the fifth day—Estimation of urea during crisis—Short convalescence.

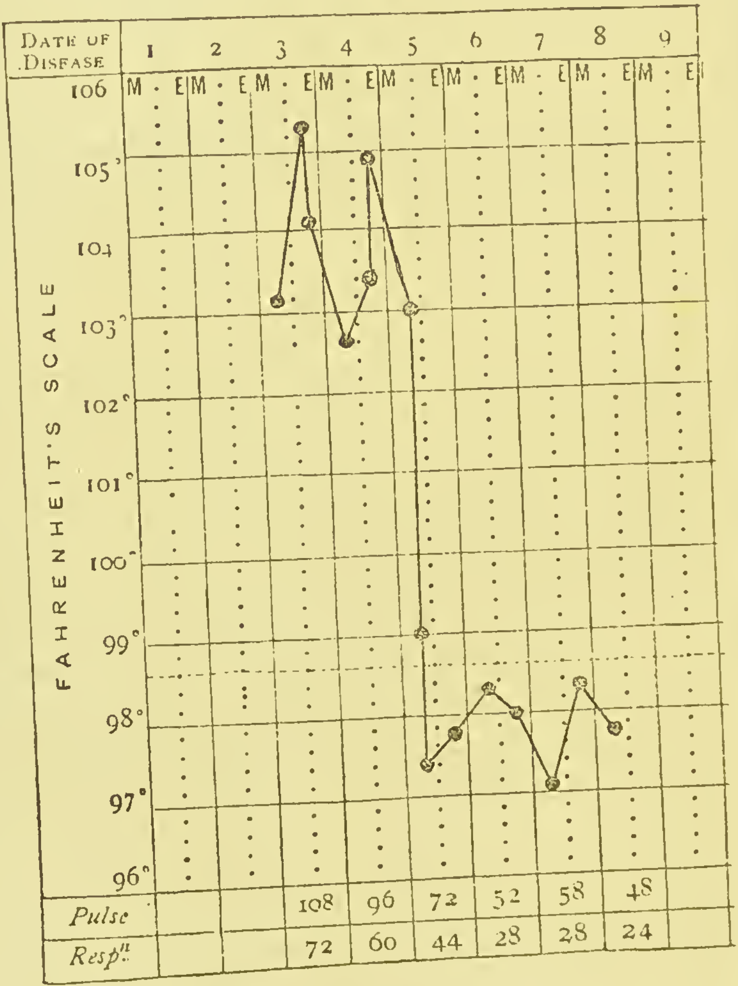
George W., twenty-one, labourer, admitted to Burdett Ward, Westminster Hospital, June 7, 1875.

A healthy, stout-built man, addicted to some excess in beer, till within a few months, when he became temperate. He was at work and well till the evening of June 3, when he went to bed and slept as usual. At six o'clock the following morning he complained of stiffness, pain in his legs, and headache ; he had a slight rigor and vomited. Within half-an-hour of the commencement of these symptoms, cough set in, and along with it blood-coloured expectoration. He had also pain in the right side, but this did not appear to be acute.

When seen on the third day of his illness, he had a flushed, anxious face, and lay, not evenly on his back, but with the left or affected side raised above the level of the other, a position which he maintained throughout. Breathing was extremely rapid and panting, the nostrils being kept in rapid action. With a pulse of 108 only, and no marked 'stitch,' the respirations reached 72. Cough was short, not harassing, sputa scanty, and of the characteristic rust-colour. Over the upper half or less of the right lung the respiration was bronchial, approaching tubular ; there was diminished resonance posteriorly, *and no crepitus.* The tongue was moist and clean, but the man had no desire for food. He did not now complain of pain.

On the morning of the day of admission (being the third of illness) the temperature was 103·2, but in the evening it rose to 105·2, falling again towards midnight to 104° (Chart 8). On the following day (June 8) the physical signs were more marked anteriorly, the infra-clavicular region yielding marked dulness, and tubular breathing audible both there and posteriorly, with other phenomena of solid lung. The patient's aspect was unchanged; he had wandered

CHART 8.



	1	2	3	4	5	6	7	8	9
Pulse			108	96	72	52	58	48	
*Resp*n.			72	60	44	28	28	24	

in mind through the night, but had no active delirium. The motions were dark and rather loose, sputa still rust-coloured ; urine contained only a trace of chlorides, was free from albumen. *The pulse was* 96 *and the respirations* 60. The morning temperature of this day was 102·8 ; in the afternoon it had risen to 103·3, and the same night to 105°.

At the commencement of this second temperature rise (that is to say, at two o'clock in the afternoon of the fourth day of pyrexia), it was directed that the urine passed during the next twenty-four hours should be collected for quantitative estimation [1] of the urea eliminated. [It will be seen directly that in the course of this collection crisis took place.] The total urine so passed (none having been lost) was 1,130 cubic centimetres, of specific gravity 1020, percentage of urea 3. The total amount of urea eliminated in the twenty-four hours 33·9 grammes (a quantity hardly above the normal). Of chlorine excessively minute trace only.

Meanwhile the patient fell into a deep sleep, which was broken only once or twice when he roused himself to pass water. On the following morning, when still in a state of lethargy, the pulse was 72, respirations 44. The temperature in the early morning was 103°, or just two degrees lower than the previous night, and continued rapidly falling. In the forenoon it had sunk to 99°, and by mid-day had subsided to 97·3, or more than a degree below the line of normal heat. In other words this man's critical change, which concurred with a normal urea discharge, was accompanied by *a fall in temperature, within little more than twelve hours, of more than seven degrees Fahrenheit.* With these marked phenomena the aspect of the patient corresponded ; and the sudden change from the fever flush and anxious, watchful aspect to an expression of mere drowsiness from loss of sleep was very striking.

On the 10th the sputa were quite uncoloured, watery and spittle-like ; respiration at the upper part of the right lung was still bronchial, and some coarse (redux) crepitation was now heard for the first time. The man was now drowsy and contented. The temperature had risen, but was still slightly below the normal point ; pulse was 52, respirations 28, appetite returning. For a day the pulse was below 50. In ten days he was up. Treatment there was none. The man took no alcohol until his convalescence was established, and then only a daily glass of beer. The mixture of acetate of ammonia formally ordered was never rigidly insisted on, and soon discontinued. He hardly lost flesh in the attack, and it was not necessary to seek to hasten his recovery with any medicinal appliance.

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[1] For the greater assurance of accuracy, Dr. Dupré, F.R.S., lecturer on chemistry at the Westminster Hospital school, was kind enough to undertake this analysis.

The case shows that early high temperature does not imply prolonged or dangerous attack; that the affection of the upper lobe has not necessarily special phenomena of its own; that the amount of urea elimination is not necessarily either in excess or defect at the time of crisis. It also shows from the temperature chart that the ratio between pulse and respiration varies considerably from day to day, and that a quickened respiration may correspond with a lowered pulse and lowered temperature.

The final issue suggests certain reflections in the matter of treatment. What if this man, when breathing sixty a minute, had been bled? What if alcohol had been given at the same point, or large doses of quinia? Could the bleeding or the drugs have escaped the credit they would not deserve?

Case IV

Pneumonia of left apex—Wild delirium—Insensibility—Sudden amendment on the ninth day—Rapid recovery.

Elizabeth T., twenty-nine, married woman, with no phthisical history in her family and herself hitherto perfectly healthy, was seized with violent ear-ache on July 29 at 8 A.M., having arisen feeling quite well. In three hours some discharge commenced from the right ear, with immediate relief to that pain, although general headache continued. The same evening pain commenced in the limbs and left side. She was unable to lie on this side, or to take a deep breath. On August 2 slight shivering occurred, and she was admitted into hospital the same afternoon (the fifth day of illness).

The pulse was now 152, small, and respirations 48; the temperature 104·2. On this day no abnormal sounds could be detected either in lung or pleura. On the next morning, however, breathing was tubular at the left apex, with dulness on percussion anteriorly reaching to the third interspace. There was also a little fine crepitation. Slight cough, with sputa viscid and rusty, pulse 144, respirations 48. In the urine was no albumen, but marked deficiency of chlorides.

It is not necessary to enter here particulars of the progress of the physical and other signs for the two following days. It is sufficient to say that consolidation of the upper portion of the left lung was very clearly indicated, that the pulse remained over 140, with respirations varying from 50 to 72, and temperature a little over or a little under 104°, the patient being quiet and not delirious, her tongue moist and furred. She was given ammonia and chloric ether, and three ounces of brandy daily.

On the morning of August 5 (being the eighth day of illness) this woman became somewhat delirious; her pulse was at this time 146, and respirations 72; the area of dulness had not extended, and coarse inspiratory ('redux'?) crepitation was now heard. As the day drew on the delirium deepened, and in the afternoon the patient began to rave, throwing her arms about, beating the bed, refusing all nourishment, and wasting her little strength in piercing yells. The pulse was now flickering, and hardly to be counted; the respirations were no less than 100; the eyes became fixed and upturned, with *total insensibility of the conjunctiva*. The features were now pinched; tongue and skin dry and parched; death seemed impending. With great pains she had been got to swallow by drops nearly half an ounce of brandy. She was now—when seemingly dying—ordered an enema of eight ounces of beef-tea with half an ounce of brandy, only a part of which was retained. Upon this some improvement appeared in pulse and feature, but delirium of the same wild kind as before was not absent for long. The enemata were repeated at intervals. Towards midnight the respirations varied between 72 and 90, and the pulse improved in strength. Owing to the patient's violence and restlessness it was impossible to take the temperature accurately. On the evening of the 4th it was 104°, and it is believed that it did not afterwards exceed that point.

Fifteen minutes after midnight on August 6 (the beginning of the ninth day of illness) this woman suddenly recovered her senses and recognised her husband. She now accepted nourishment readily. The pulse fell to 120, respirations to 60, and temperature to 100°; a fall of over 3° from the previous day. The urine, which had been retained for some hours, was now passed in large quantity. When left to herself the patient would fall into a muttering delirium, from which she was easily roused. The skin was now perspiring. She shortly fell into a sound sleep, and with this it may be said that her peril ended. Later on in the morning of the 6th, when the pulse was 124 and very feeble, and respirations 44, the body freely sweating, the temperature was still 100°; it fell *continuously* throughout the day, and was 99° on the morning of the 7th. The sputa were now yellowish and without blood-stain. Signs of consolidation of lung were still marked. On the 7th some redux crepitation was heard, and by the 9th dulness had almost disappeared.

It is unnecessary to pursue the case in detail. After the 8th the temperature remained normal; the pulse and respiration fell together, although the former remained over 100 until August 10. On the 16th, being just three weeks from the earliest symptoms, and ten days from the crisis, the woman was able to get up for a while, and shortly after she went out well.

This case suggests reflections upon treatment similar to the one preceding.[1] It is also an illustration of pneumonia of great severity, and with profound implication of the nervous system, yet locally of no great extent and soon reaching its term. The gravity of the case is by no means measured by the lung inflammation.

CASE V

Pneumonia of upper lobe—Active delirium—Late appearance of physical signs—Lividity and symptoms o collapse on the ninth day along with defervescence—Crisis on the tenth day.

John A., aged seventeen, admitted March 19, 1875, having been taken ill on the evening of the 17th with shivering, giddiness, and nausea, and delirious during the same night. When examined on the 20th he had a pulse of 130, and temperature 104·6; tongue coated, his condition generally resembling enteric fever. He wandered when left to himself, but answered questions rationally. The chest-sounds were noted as normal. The same night he became actively delirious, and some *fine crepitation was now first audible* (without dulness) over the upper third of the right chest. On the 21st (or fourth day of illness) the physical signs were more marked; dulness at the right apex, tubular respiration, the heart's sound being transmitted with great clearness through the solid lung. He had now rusty sputa. During the next three days, from March 22 to 25 (the eighth day of illness) the patient continued violently delirious; temperature 103·6, rising (on the 24th) to 105·2; pulse 130, full and hard; respirations from 68 to 76.

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[1] Besides the above, a case illustrating the extreme difficulty of discriminating between the natural course of the disease and the effect of remedies will be found in the chapter devoted to Treatment (Chap. XX).

On the 26th (the ninth day) with a falling temperature (101°), and pulse still 130, he was, as to his general condition, markedly worse; his face was now livid and pupils dilated, the tongue was dry and parched, and he sweated profusely. No urine had been passed during many hours, and the accumulation in the bladder had to be withdrawn by catheter. The violent delirium had given place to mere prostration. He had profuse sweating, was barely conscious, and the motions were passed in bed; he appeared to be dying. He had been given four ounces of brandy from the first; the quantity was now doubled, and the ordinary means of stimulation were employed.

At 3 A.M. of the 27th, having passed water naturally some hours before, and had some diarrhœa, this youth fell into a deep sleep (which the nurse believed to be his last); he awoke, however, at 9 A.M. greatly improved, with a temperature 99·4, clean and moist tongue, and sputa but slightly blood-tinged. The pulse was now 112, and respirations 50. Two days from this the pulse was 84, while the respirations were 32. The patient had no bad symptom from this time, and was convalescent a week later.[1]

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[1] To the two cases above related, tending to show a special implication on the part of the nervous system sometimes characterising apex pneumonia, the following may be added. It was originally quoted by one of us in the 'St. George's Hospital Reports,' vol. i. p. 343.

The patient was a nurse-maid, aged twenty-eight. She had been taken suddenly ill with rigor, cough, and some dyspnœa six days before, and her friends had noticed at the same time a strangeness in her manner, but no actual delirium. There was nothing in the aspect of the patient (unless labial herpes be so considered) to indicate the nature of her disease. There was found, however, on percussion, marked dulness below the right clavicle, with which corresponded tubular breathing and brassy ring of the voice (temperature is not recorded); the pulse was 112.

On the morning following her admission this girl sprang out of bed and ran naked through the ward. From that time, for four days, she continued actively delirious, making grimaces and talking wild nonsense. At the end of the fourth day (the usual lung symptoms remaining in abeyance) signs of sinking appeared; the tongue began to get dry, and sordes formed about the mouth. But now, of a sudden, the pulse, which had hitherto been 112 or thereabouts, fell to 70, and its rate did not afterwards exceed 80. Meanwhile the active delirium subsided, and the woman fell into a bewildered, puzzled state of mind, as of one just awakened. From this she very gradually returned to her natural manner. At the same time

CASE VI

Apex pneumonia—Delirium throughout—Crisis on seventh day, followed by transient melancholia.

Fred. S., æt. twenty-one, a draper's assistant, admitted into the Middlesex Hospital on January 14, 1888, having been attacked five days before (Jan. 9) with pain in the right side, vomiting and pain in the stomach. He became feverish and delirious on the 13th, when he left his bed, got out of window and walked down the street partially dressed. He is said to have had 'congestion of the lungs' when sixteen years of age.

On admission he was seen to be well-nourished, muscular, but prostrate, and was delirious. T. 103·8. P. 120, regular, good volume. R. 50. Lips dry and cracked, livid; tongue tremulous, coated. *Alæ nasi* working. There was dulness on percussion over the upper lobe of right lung, in front,—to level of fourth rib, behind, —in supraspinous fossa. High-pitched tubular breathing over the dull area, weak breathing elsewhere; fine râles over lower lobe, and also in right axillary region. Heart sounds weak. Abdomen distended.

An ice-bag was applied to the upper part of right chest, and a mixture containing bromide of potassium and carbonate of ammonia prescribed.

January 15 (*seventh day*).—The maximum temperature was 100·4 at 10 A.M.; at 8 P.M. only 98·6. P. 108; R. 48. Did not sleep, but was very restless and delirious during the night. Lips more cyanotic. Tongue dry and coated. The area of dulness has extended behind over the greater part of scapula (thus coming to involve the apex of the lower lobe as well as upper lobe), and tubular breathing still marked. There is also a small area of tubular breathing in the right axilla. Elsewhere harsh breathing. Involuntary micturition.

In spite of a chloral and bromide draught, and the hypodermic injection of hyoscyamine, there was no abatement in the delirium.

January 16 (*eighth day*).—Temperature throughout normal

the disappearance of the morbid sounds at the lung's apex, and their replacement by perfectly healthy breathing, occurred in remarkable harmony with the general progress towards health. The patient was finally discharged in all respects well.

Examples of hallucination and altered mental state among the symptoms of pneumonia (not necessarily of the apex) will be found in the text (p. 48). Acute mania is sometimes a sequel.

and subnormal. Still no sleep; constant muttering delirium and picking of bedclothes. Hands tremulous. Face flushed. Lips livid, dry; tongue dry. The dulness and tubular breathing still present, but less marked. Urine contains a trace of albumen.

January 17 (*ninth day*).—Refuses food, and resists examination. Constant muttering delirium. Still no sleep. Pulse 90, weak. The temperature rose to 100° in evening. Stimulant given.

January 18 (*tenth day*).—Continues in a melancholic state, and last evening had to be fed forcibly. During the day, however, his mental state somewhat improved, and he took some food without resistance. But there is still some delirium. The right upper lobe is dull on percussion; breathing weak. Fine râles at left base.

January 19 (*eleventh day*).—Has slept a little at intervals, and is no longer delirious. Dulness at right apex clearing up, except posteriorly, where there is marked bronchophony. Breath sounds everywhere weak. Fine moist râles at left base.

He now continued to rapidly improve; and on leaving the hospital on February 11 examination showed slight impairment of expansion of right apex, with weak breathing, and slightly increased vocal resonance, but no dulness.

This case affords another illustration of marked mental disturbance, which commenced two days before the crisis, when it became more profound, the delirium and insomnia passing into a state of transient melancholia. The association of these symptoms with pneumonia of the upper lobe is again illustrated.

CASE VII

Double pneumonia—Persistent physical signs—Remittent pyrexia—No evidence of tubercle.

Mary Ann C., twenty-four, maid-servant, admitted into the Middlesex Hospital on May 2, 1887. She said that she had caught a cold a month ago, which was followed by difficulty in breathing and pain in the right shoulder passing round to the back. A week ago, on April 28, she became worse, having pain and swelling in the right ear, and also fresh pain in the right side with more dyspnœa and fever.

On admission, T. 103·6. P. 132. R. 60. Cheeks flushed, lips dry, tongue coated. Skin hot, breathing rapid and painful. There was impaired resonance at the right apex, and dulness over both backs from the angle of scapula downwards. It was only on the right side that there was any tubular breathing, which was limited to the area of dulness. There were moist râles over the whole chest.

Ether and ammonia mixture was prescribed, and three ounces of brandy.

On the 3rd the dulness was found to extend over the lower two-thirds of the right back, where the breath sounds were tubular, and where there were some dry fine râles with inspiration. The urine was found to be deficient in chlorides. She took nourishment well.

On the 7th the pulse was stronger, 98. She seemed better. Wine substituted for brandy.

On the 8th there still persisted dulness and tubular breathing at the right base, and now it was noticed that the breath sound had a like quality at the left base, which had throughout been dull on percussion.

A mixture of bark and ammonia was ordered.

On the 13th it is noted that there was high-pitched tubular breathing and fine crepitation over both backs.

The temperature from May 2 to May 4 had ranged from 102° (morning) to 104·6 (evening); from the 5th to the 12th from 98° (morning) to 101° (evening).

The cough now became more troublesome, and some bronchial breathing could be heard in front at the lower part of the left lung. The expectoration was scanty, muco-purulent.

On the 20th it was noticed that there was more expansile movement of the left side of chest than of the right. The right back was dull throughout; vocal fremitus abolished, but her voice was weak. In supraspinous fossa breathing vesicular; in infraspinous fossa fine crackling râles; at angle of scapula intense bronchial breathing, and abundant fine crepitation thence to base. Over the left lung behind the resonance was normal in scapular regions, but dull below, whilst there was intense bronchial breathing with crepitation as on the other side. In front the breath sound was harsh with prolonged expiration at both apices. There was constipation.

On the 26th it is stated that she had been sweating lately. She complained of pain in right side of chest, which was relieved by poultices. The sputum was muco-purulent, no bacilli could be found in it.

The temperature from the 13th to the 26th ranged from 99° (morning) to 102° (evening).

On the 31st the breathing over the scapula and in the interscapular region was harsh, râles scanty; but there was still high-pitched bronchial breathing, with bronchophony and pectoriloquy at the angle of scapula. The cough was less troublesome.

On June 3 it was noted that the dulness does not extend so far up the left back, but persists unchanged on the right back.

Three grains of iodide of potassium were added to the ammonia and bark mixture.

On the 10th there was still intense bronchial breathing in left interscapular region, with abundant coarse metallic râles. Cough again troublesome; sputa viscid, muco-purulent.

During the next week the expectoration increased somewhat in amount, and once was streaked with blood. But the patient improved in general condition, and on the 17th was allowed out in the garden with benefit.

The last material note prior to her leaving the hospital was on June 27. Then the right back was absolutely dull from mid-scapula to the base; the dulness over the left back limited to the extreme base. The breathing over the right lung was no longer bronchial, but weak, and some fine crepitation (friction?) could be heard posteriorly. At the left base the breath sounds were harsh and blowing, with moist crepitation. In front the percussion note was almost tympanitic on the left side, normal on the right. There was still some muco-purulent expectoration.

The temperature from May 27 to June 28 ranged from 98° (morning) to 101° (evening), but it had become far more irregular than previously.

The foregoing case—of which the record has been considerably abbreviated—was regarded rather as an example of chronic pneumonia than of the acute disease, and yet the pyrexia was considerable (markedly remittent in type), and the signs of consolidation on both sides—particularly the left, which was at no time so much involved as the right—gradually cleared up. Unfortunately, no opportunity has been given for a physical examination subsequent to June 28, 1887, when she left the hospital. We should be inclined to regard the case as one where the pneumonia was associated with considerable pleurisy, which may account for the persistence of the extensive area of dulness on the right side.

Case VIII

Double pneumonia, of insidious onset—Two attacks within one year, each marked by intermittent pyrexia.

Emma H., twenty-nine, married, admitted into the Middlesex Hospital, under the care of Dr. Douglas Powell, on December 24, 1885. Her father was asthmatic, and one of her three sisters suffers from 'chest complaint.' Her illness began insidiously about a fortnight after her confinement (the child only lived a week) in October, when she began to suffer from pain in the right inframammary and axillary regions, to lose flesh and appetite, and to have night-sweating. She had become much worse during the past fortnight.

She was a thin, ill-nourished woman, and the signs were suggestive of her having had right pleurisy, for this side of the chest was retracted, and there was pain in the axilla, where friction was audible, mingled with râles that attend on coughing. There was dulness in the infra-axillary and infra-scapular regions. She had a paroxysmal cough. There was moderate pyrexia. (100° to 101·6.)

On December 30 she experienced occasional sharp pain in the *left* lower axilla, and on this day there was a sudden accession of fever, the temperature rising to 104·4. P. 144. R. 36. On examination the resonance over the left lower lobe was found to be impaired, the breath sound weak, except near the scapular angle, where it had a tubular quality, and where some subcrepitant râles were audible.

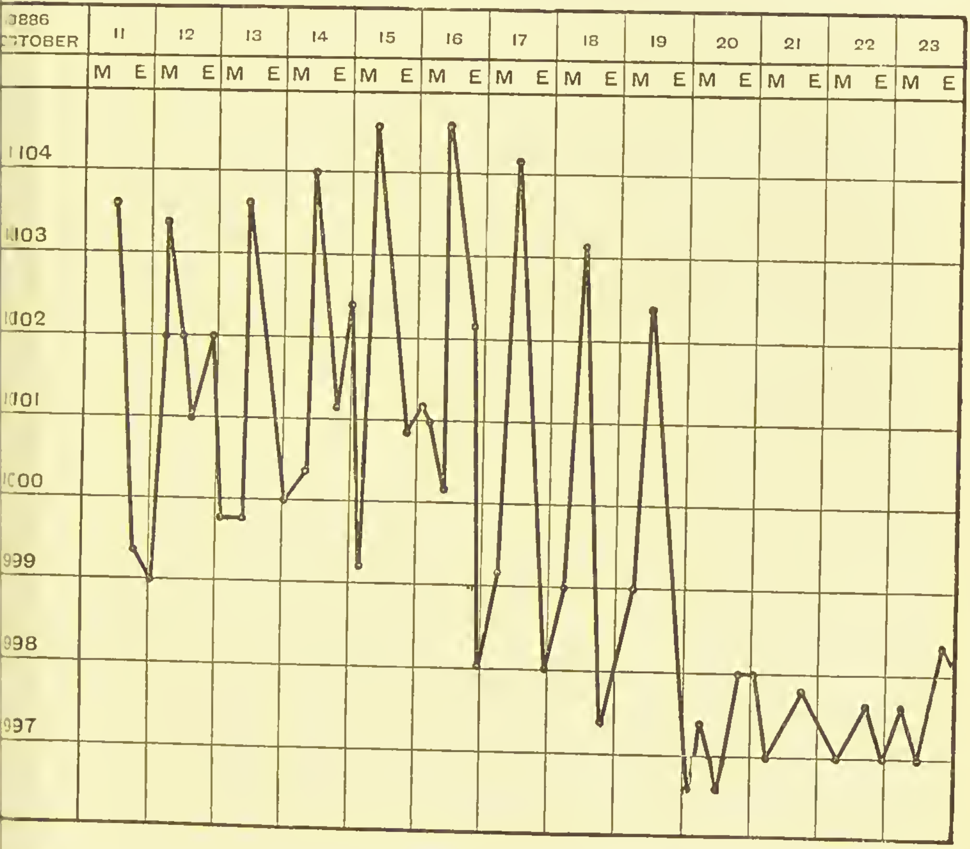
On the 31st, the morning temperature was 104°, evening 101·6.

The pyrexia was most irregular, and may be best given in a tabular form.

—	2 A.M.	6 A.M.	10 A.M.	2 P.M.	6 P.M.	10 P.M.
Dec. 30, 1885	—	101·6	—	102·8	103·2	104·4
,, 31 ,,	—	104·	104·4	104·	103·4	101·6
Jan. 1, 1886	99·6	101·6	102·8	103·8	102·4	101·4
,, 2 ,,	99·6	100·	104·6	103·	101·2	100·
,, 3 ,,	99·4	99·	104·	—	100·6	99·4
,, 4 ,,	99·	98·6	—	101·6	99·	—
,, 5 ,,	99·4	100·6	102·6	100·2	98·4	97·6
,, 6 ,,	97·8	98·4	101·6	98·2	98·	98·
,, 7 ,,	98·4	98·6	98·2	—	97·6	97·6

These figures are given in order that a comparison may be made with the type of pyrexia which the same patient exhibited during a similar pneumonic attack some months later. As regards the signs of the present attack in the left lung, they became more plainly developed over the greater part of the lower lobe on January 1 and 2, but after that rather rapidly declined, and by the 11th had almost entirely cleared up. The pulse rate, which on December 30 and 31 and January 1 had been as high as 144, fell to 102 on the 2nd, and 96 on the 3rd.

CHART 9.



On October 11, 1886, the same patient was again admitted into the hospital, this time under the care of Dr. Coupland. She had kept well in the interval up to two weeks before her re-admission, when she began to suffer from a cough and to feel very weak.

When admitted: T. 103·4. P. 144. R. 36. Cheeks flushed. Lips dry. Skin hot and dry. Some cough, with bronchitic sputum. Complaining of pain in the right chest. Expansion of this side defective. Resonance in all parts except over the *right* back from angle of scapula to base. Here the vocal fremitus and resonance are increased, and there is tubular breathing, together with fine crepitation and friction sounds. Elsewhere the breathing is harsh, with sibilant rhonchi and coarse mucous râles. Heart sounds normal. Tongue coated on dorsum; margins red. Constipation. Urine normal.

It is noteworthy that on this, as on the previous occasion, signs of consolidation began to appear in the left lower lobe. On October 13 it is noted that the dulness over right back is less, and the breathing is no longer tubular; and coarse râles have replaced the finer crepitation. At the same time it is noted that dulness and tubular breathing have appeared over the base of the *left* lung. However, it seems from a subsequent note (October 16) that the signs of consolidation returned to the right back, suggestive of the supervention of pleuritic effusion; whilst on the left side, over the lower one-third the breath sound was intensely bronchial and vocal resonance increased. A puncture with a hypodermic syringe in the left back confirmed the diagnosis of effusion at this base.

The course of the pyrexia was even more markedly remittent than on the previous occasion (see Chart 9).

—	2 A.M.	6 A.M.	10 A.M.	2 P.M.	6 P.M.	10 P.M.
Oct. 12, 1886	98·	99·8	103·6	101.	101·8	102·
,, 13 ,,	99·8	99·8	101·	103·4	102·6	102·4
,, 14 ,,	100·	100·4	104·	103·	101·2	102·2
,, 15 ,,	101·	99·4	104·6	103·6	101·8	101·2
,, 16 ,,	101·	100·4	104·6	102·2	100·	98·
,, 17 ,,	98·6	99·2	104·2	101·8	99·	98·
,, 18 ,,	98·4	99·	103·2	100·	99·	97·2
,, 19 ,,	98·6	99·	102·6	100·6	96·4	97·
,, 20 ,,	97·4	96·	97·	98·	97·	98·
,, 21 ,,	98·	97·	97·	97·2	99·8	97·4

It will be seen that on October 19 there was a definite crisis, but the tubular breathing did not disappear until the 21st, and it was fully a fortnight before the râles cleared up, whilst the dulness at both bases persisted, probably from the existence of pleural thickening.

The severity of the constitutional disturbance, added to the physical signs, compelled a diagnosis of pneumonia—double basic—rather than pleurisy alone. And yet the case was very anomalous. It was insidious in its onset—remarkable for the entire absence of rusty expectoration from first to last,—and for the singular type of pyrexia. The latter is not, however, unique. Wunderlich notes that the course of acute pneumonia may be interrupted by one or two intercurrent falls of several degrees; and Dr. Wilson Fox speaks of cases of an intermittent type apart from malaria where there are apyrexial periods of 12 to 36 hours, the exacerbations appearing to be due to an irregular progress of the pneumonia (see p. 203). In the present case there was no malarial influence at work, but the patient was a debilitated woman, and we can only conclude that the character of her pulmonary affection and of the pyrexia accompanying it must have been in some way related to her individual constitution.

Case IX

Pneumonia—Hæmoptysis—Chlorides not diminished—Slow pulse—Crisis on the sixth day—Bronchitis supervening—Convalescence accidentally delayed.

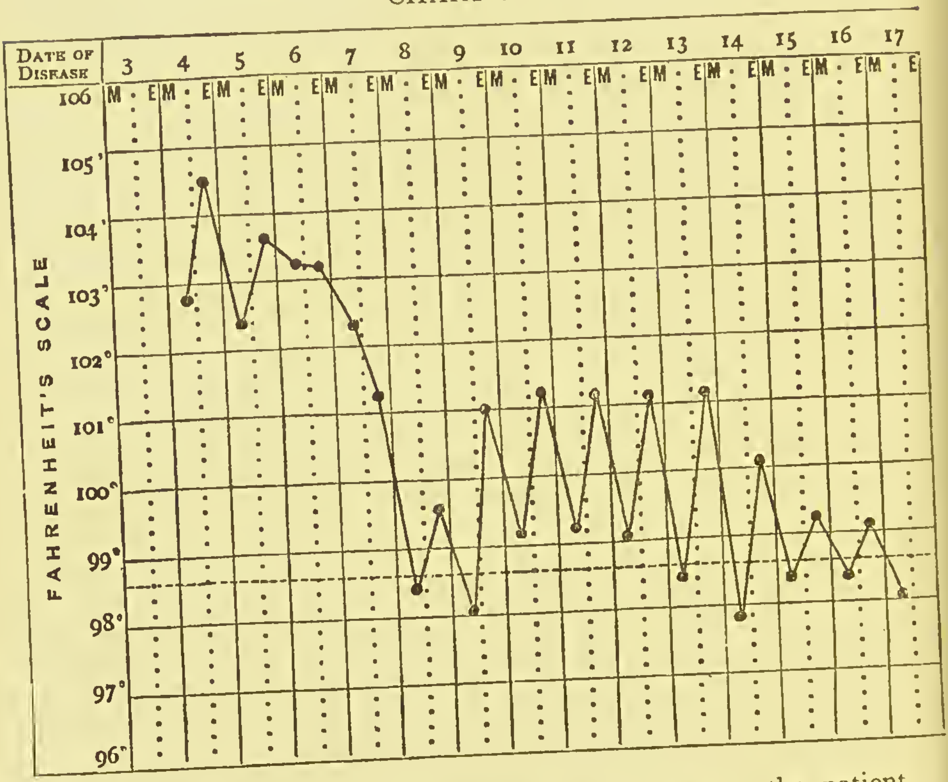
Henry B., aged twenty-four, lighterman, admitted July 1, 1875, into the Westminster Hospital. He had had rheumatic fever ten years before, uninterrupted health since, and is of temperate habits. He was quite well up to and including June 27 (Sunday), on which day, in thinner clothing than his working-dress, he took an excursion into the country. (The season was ungenial, and after much enquiry this change of clothes had to stand for cause of the illness which immediately followed it.) Early in the morning of the 28th, after a good night, he was seized with pain across the right chest and in the forehead, but without shivering; he managed to get through two hours' work and then gave in, fairly beaten. The same evening he spat up 'congealed blood.'

On admission, early on the fourth day of illness, his face was flushed, aspect anxious, decubitus dorsal; the *alæ nasi* dilating considerably with respiration; he was without herpes; the pulse was full, soft, and 92, respirations 46, temperature 102·8. The tongue was moist and thickly furred; the sputum was rather red than rusty, and of quite fluid consistence. Over the base of the right lung, up to about the fifth rib, was tubular breathing, crackling crepitus, and increased voice resonance.

The same evening at 11 P.M. pulse was 84 and respirations 54. The temperature had risen to 104·5, its highest point.

On the following day (fifth of illness) pulse was 88 and dicrotous, the respirations 56, temperature 102·5; a slight herpetic eruption had appeared on the upper lip. In aspect the patient was little changed, the face being still much flushed. The expectoration was watery and blood-stained, the urine had a specific gravity of 1025, was without albumen, and showed abundant chlorides; the tubular breathing remained, with less crepitation. That evening the pulse was 88, respirations 48.

CHART 10.



On July 3, the sixth day of illness, he was found perspiring freely, the sputum was more viscid, the morning temperature was 103·2, the evening perceptibly *lower*, 103·1. Both by these phenomena, and still more in his altered aspect, was this day marked as the crisis of the fever. On the following day, the temperature continuing to fall, insomuch that the evening temperature was a degree lower than that of the morning, pulse was 90 and respirations 42. On the 5th, or eighth day of illness, redux crepitation was audible over the base of the affected lung. The pulse had fallen to 64, but the respirations were 44. The temperature was now normal. On the following day, and for the next four, bronchitis was developed ; rhonchus became audible over both lungs, the sputum became more abundant, but now colourless. The evening temperature was pretty constant at a little over 101°, the morning a little over 99°. The cough had changed its character, and was more troublesome and constant. On the 10th, however, these symptoms began to subside, and now the resonance of the right lung was restored, there remained no small or medium crepitation, and no trace of tubular respiration. On the 14th the man got up ; he was convalescent on the 21st, and on the 26th he went out recovered.

In the course of the fever the man had exceptional wasting, but rapidly recovered flesh so soon as this had subsided. There was no reason to suspect structural lung disease.

This case has some unusual features. There was no rigor ; the sputum was never of the characteristic rust colour—at first it was blood, and later a blood-tinged watery secretion, with but little viscidity ; the pulse was never frequent, and with a respiration quickening from 46 to 54, it fell from 92 (its highest frequency) to 84 ; the chlorides were never deficient ; bronchitis interrupted recovery.

Yet, with these accidents, it was pneumonia and nothing else. In aspect, temperature, defervescence, physical signs, as well as in the mode of recovery on the part of the lung, it might even be called typical. The persistence of the chlorides in the urine, along with the unusual character of the sputa, is of interest in reference to the hypothesis of Dr. Beale, which connects hepatisation with the disappearance of these salts.

The infrequency of the pulse when there was high pyrexia and notable dyspnœa, is seen from time to time both in pneumonia and the continued fevers.

As for the relation between the bronchitis and the pneumonia it was accidental. The patient's bed was somewhat exposed to draught, the wind in the east and temperature remarkably low for the season. So the man took cold, as any other might. It is especially to be observed that this catching cold *in no way retarded the progress of the lung towards recovery.* The bronchitis arose quite separately, as the pneumonia was departing ; it persisted for about four days, while the lung was undergoing resolution, and this process was not arrested by its occurrence.

To the foregoing narratives of pneumonia, severe and threatening, yet in every instance recovering rapidly so soon as the disease 'took the turn,' may be appended in brief outline, lest the fatality of the disease should be underrated, the circumstances of a case which without much warning took a fatal course, death occurring when a favourable 'critical' change might have been expected.

CASE X

Pneumonia fatal within ten days—Highest temperature 103·2—Commencing disease of kidney.

Thomas G., thirty-nine, carman, admitted into Westminster Hospital November 18, 1873, was suspected of intemperate habits, but believed to be in perfect health until six days ago, when he was seized with rigor. He has now a flushed face, with herpes on the lips. Tongue is brown and dry, pulse 130, weak and compressible ; respirations 44, temperature 103·2. Over nearly the whole of the right lung respiration is tubular, with corresponding percussion dulness and some fine crepitation ; there is no albumen in the urine, and only a trace of chlorides. He is put upon beef-tea, and given ammonia in doses to stimulate, with three ounces of brandy daily.

The following day (the seventh from the rigor) the skin is warm and moist, and tongue less dry; he has had some sleep, the pulse is 140 and less compressible, temperature is 102°, and respirations 40; the physical signs as yesterday, save that crepitation extends higher up.

On the 20th his condition was one of great prostration, the pulse somewhat more frequent; other symptoms the same. On the 21st (the ninth day), the temperature being 102·4, pulse 138, and respirations 46, he exhibited all the signs of approaching death, which arrived some hours after midnight.

Post-mortem Examination, *November* 22, 1873.—Body well nourished.

The left lung is crepitant throughout, much congested posteriorly, and very œdematous, a large amount of serum escaping on section; adhesions at base of this lung; no fluid. The bronchial mucous membrane markedly thickened, tough and fibrous.

The right lung is universally adherent, a small amount of coagulated lymph on pleura; as to the upper third of its upper lobe it is intensely hyperæmic, yet crepitant. Elsewhere this lung is completely solid; its cut surface is granular. The consolidation is grey, and most perfect as to the lower lobe. Between this and the hyperæmic apex mentioned above is a portion intermediate between hyperæmia and absolute solidity.

The heart normal as to size and thickness of walls, whose texture is very firm; there is much sub-pericardial fat. The left ventricle is firmly contracted. In all the other chambers are firm (post-mortem) clots. All the valves are competent, and orifices of normal size. There is some atheroma at root of aorta; the coronary artery apertures are patent. There is a marked absence of fluid blood. The other organs call for no remark, except the kidneys. Of these the cortical substance of the left is somewhat diminished; its weight 7 oz.; of its fellow 6½ oz. The spleen is very pale.

The case illustrates the value of the pulse as a prognostic sign when the temperature is not very high nor the respirations very frequent. On the sixth day the pulse was 130, with temperature 103·2; on the day following, with some general improvement, a lower temperature and somewhat reduced rate of breathing, the pulse had risen to 140; it was by this mainly that the man's actual condition was indexed. There was no marked delirium, and the prostration, which became extreme near the time of death, was not great at an earlier period.

The organs, apart from the lungs, were mostly healthy, but there was some atheroma of the aorta, and the cortex of one kidney was diminished. The condition of the bronchial mucous membrane of the unhepatised lung indicated antecedent disease by fibroid increase. The case was one which with a healthier and more sober subject might probably have recovered.

Case XI

Pneumonia following accident, fatal six days later and within three days of its first physical signs.

Thomas P., barge-labourer, of muscular frame, not of drunken habits, aged 30, was admitted into Westminster Hospital. He had fallen between two barges when drunk and remained in wet clothes for two hours. When admitted to hospital he was dazed and confused, and in view of supposed injury was placed in a surgical ward. He speedily recovered, however, from a condition that seemed due to intoxication rather than the accident. The day following admission he was to have gone out as well, but, complaining for the first time of pain in the left side, he was transferred to the medical side. On the next, or third day from admission, when first seen by one of us, he was delirious, and temperature was 103°, but no physical signs indicating pneumonia were discoverable and the pulse was not frequent. Delirium continued and was of muttering kind, the man became drowsy and difficult to rouse, and his tongue dryish. On the fifth day tubular respiration was audible at the right apex as well as (less distinctly) at the left, where also was indistinct crepitus. The pulse was now 80 and shortly fell to 50. Temperature remained at about 103°. There was no cough, no spitting, and no complaint or appearance of pain. The rapid course of events prevented any examination of the urine. With nothing further to note the next day he died.

Thus from accident to death was six days; from delirium to death three days; from first observation of physical signs to death two days.

Post-mortem.—The upper lobe of the left lung was consolidated, the pleura adherent by not quite recent lymph. The lower lobe was congested merely. At the apex of the right lung was an area of imperfect consolidation about size of pigeon's egg. Flakes of lymph covered the pericardium. The liver had nutmeg appearance on section, kidneys were large, and both they and the liver gave faint lardaceous reaction.

The lung consolidation examined microscopically exhibited a small celled growth in alveoli, whose walls were little changed. In more advanced parts, however, the walls were thickened and pervaded by similar growth, and in some places the contents of several alveoli had coalesced, the lung structure at these small points having disappeared.

The case illustrates the rapidity with which pneumonia may prove fatal in an able-bodied man, apparently healthy, occupied up to the day of his illness in a very laborious employment. It shows the commencement of destruction of lung tissue within a very short time of the first appearance of lung inflammation, and forms a striking contrast to the usual course of pneumonia in a healthy young subject. It is also a very rare example of the disease attaching itself to *both* apices. In view of the condition of the liver and kidneys it can hardly be reckoned as simple pneumonia, and there was strong suspicion of intemperate habits. Yet the fact remains that an apparently healthy man may succumb to pneumonia in a few days with no clear evidence during life either of alcoholism or organic disease. The case teaches caution in prognosis, and especially so when bodily accident has been sustained shortly before the attack.

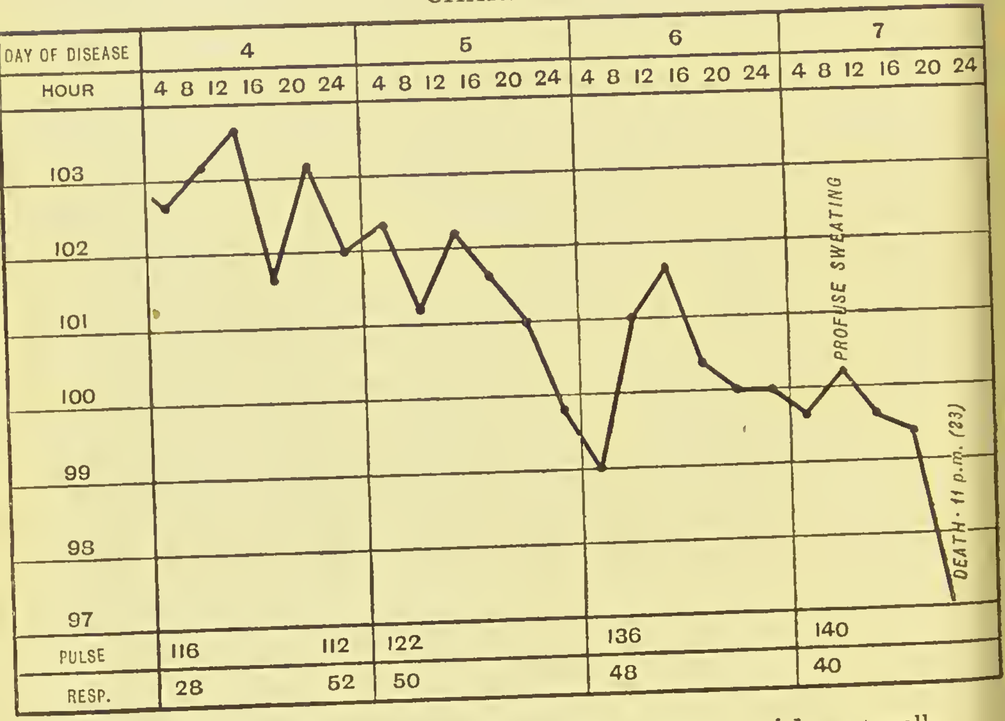
Case XII

Pneumonia spreading from its first seat—Symptoms resembling crisis—Death on seventh day—Temperature in descent from fourth day (103°) to end—Profuse sweating day before death—Much restlessness, but no delirium.

Henry C., aged forty-two, porter, admitted October 8, 1889. Father, two brothers, and a sister said to have died of phthisis. He himself has had no previous serious illness, and reports himself of not intemperate habits.

On evening of October 5, 1889, three days ago (having got wet day before), had cold shivering and headache, with stitch in right side. Two days, October 6 and 7, kept bed; went to work on 8th till afternoon, when he was admitted in following state:—Flushed, distressed face, *dry tongue*, much restlessness. P. 116. Resp. 28. Severe pain in right side temporarily relieved by leeching. Dulness and tubular breathing over right base, impaired resonance and bronchial breathing over left. A trace of albumen. The following days, 5th, 6th and 7th of disease, with increasing pulse and respiration, viz. 122, 136, 144, and 52, 50, 48 respectively, the area of consolidation increased day by day upwards (left side).

CHART 11.



Patient was sleepless and restless, but took nourishment well. Profuse sweating commenced night of the sixth day, and temperature continuing in descent, death occurred on the seventh day with subnormal temperature.

Post-mortem.—Recent lymph and fluid were found in both pleural cavities; some thin lymph also covered the pericardium; there was no endocarditis. The right lung, as regards its middle lobe and part of the upper lobe, was in a state of grey hepatisation; the left lower lobe was congested and semi-crepitant. The liver was fatty, kidneys large, their structure normal.

This case, resembling the last, probably owed its course and fatality to fatty liver, a condition not discoverable during life. It is inserted as an example of the unfavourable prognosis always attending the extension of the lung inflammation in pneumonia. It shows also how critical phenomena, such as temperature descent and profuse sweating, are fallible signs so long as there is no general improvement.

The two following cases illustrate the supervention of empyema upon acute pneumonia—a sequel more likely to occur in young children than in the adult. These patients, it will be seen, were nineteen and eighteen years of age respectively. They show that the transition from pneumonia to empyema may be quite insidious; but that the area over which bronchial breathing is heard may extend, whilst the pyrexia becomes more remittent, and the suspicion of fluid requires to be confirmed by exploratory puncture. In each of these patients it may be noticed that pleuritic signs were marked from the first.

CASE XIII

Left pleuro-pneumonia, followed by empyema—Paracentesis—Free incision—Drainage—Recovery.

Fanny Y., æt. nineteen, shop-assistant, admitted into the Middlesex Hospital on June 19, 1886. She had always enjoyed good health, but on June 11 took 'cold' and was laid up for three days. She then returned to her work, but on the 18th felt pain in the left side increased by exertion and taking breath, became feverish and had a cough. When admitted she was flushed, temperature 102·4; R. 24; P. 112; tongue coated, and over the left back from angle of scapula downwards there was dulness on percussion, bronchial breathing, broncophony, and absence of fremitus. The breath sound was accompanied by fine superficial crepitation (friction ?). The heart's apex was in the normal situation, the sounds natural. Cough not frequent; expectoration partly muco-purulent, partly rusty. On the evening of the 20th the temperature rose to 104·2. Cold compresses were applied to the left side of chest, and later temp.

103·2, ten grains of antipyrin were given, which was followed by a fall of temperature to 101·6, and free sweating. On the 21st there was a pseudo crisis, the temperature at 2 P.M. falling to 99·4, but almost at once followed by a rise to 104·2 at 6 P.M., after which it fell again to 100·2 at 2 A.M. on the 22nd. Then, for four days, it ranged between 101° and 102·5, till, on the 25th, it again reached 104°. Antipyrin was given, and there was a subsidence to 99·6 on the 27th, after which it remained about 100° to 101° during the 28th, becoming more remittent on the 29th.

As regards the physical signs during this period it may be said that at first they were those of consolidation, and that on the 22nd and 23rd, after attacks of pain in the left side requiring leeching and hypodermic injection of morphia, definite friction became audible. Then the bronchial breathing became more distant; the sputa, too, lost their rusty tint.

On June 29 the physical signs of pleural effusion in the left chest were very marked, the dulness extending up to the third rib in front, above which there was Skodaic resonance. Breath sounds were absent over the dull area and friction had disappeared. Next day aspiration attempted in axilla (sixth space) evacuated only two or three ounces of fluid; but a second attempt below angle of scapula was followed by the escape of sixteen ounces. The fluid was opaque and greenish-yellow, but not distinctly purulent.

There was, however, no marked abatement of the pyrexia, and the signs of effusion returned. On July 13 a trace of albumen appeared in the urine, and on this day an exploratory puncture below angle of scapula resulted in the detection of pus.

Accordingly on the 14th Mr. Gould made a free incision in the mid-axilla, removed an inch of rib, and gave vent to about a pint of yellow pus and flakes of lymph; a drainage tube was inserted.

The good effect of this procedure was seen in the fall of the temperature to 97·8, and with but few exceptions the temperature remained henceforth normal. The patient did well, gaining flesh and strength, although the urine continued to yield a trace of albumen until August 13. The wound closed on September 13, and before her discharge on the 17th there remained only some impaired expansion of the left side of chest, and impaired resonance at the base of lung, but breath sound was audible everywhere.

Case XIV

Pleuro-pneumonia (left base), followed by empyema—Paracentesis—Free incision—Drainage—Recovery.

Walter G., eighteen, a page boy, admitted into the Middlesex Hospital on May 9, 1885. Three days before he complained of pains in the back, and on the morning of the 7th was attacked with shivering and severe catching pain in the left side. His breathing was quick (52) and shallow, alæ nasi working, cheeks flushed, temperature 101·8, P. 132, tongue coated, skin slightly moist. There was dulness on percussion over the left front and axilla, from nipple level downwards, and behind from the level of the spine of the scapula. Fine crepitant râles and weak breathing were alone audible over this area, where vocal resonance was exaggerated, but the fremitus abolished. The percussion note over the right lung was normal, except at the extreme base, where it was slightly impaired, and where also occasional crepitation was audible. Heart's apex beat in normal position, sounds natural. Urine free from albumen. In the evening the temperature rose to 103·4.

Next day tubular breathing was audible at the angle of the left scapula. The expectoration, too, was stained a faint red tint.

Cold compresses were applied to the left side.

May 11.—Expectoration rusty. Pain in left side less. Temperature fell to 99°. Fine crackling râles (? pleuritic) over the lower part of left front and axilla. No change in tubular breathing or dulness. These signs persisted with but little change for a few days, except that on the 14th it was noticed that the tubular breathing could be heard over the whole of the dull area, in front as well as behind, and that vocal fremitus was absent over the same area. The pyrexia, too, did not abate.

May 15.—Marked impulse in fourth and fifth spaces to right of sternum. Extension of dulness upwards on left front to level of third rib. Absence of vocal fremitus, and tubular breathing as before. Ægophony and pectoriloquy.

May 16.—Temperature 99·6. Paracentesis by aspirator and syphon in left fifth interspace, mid axilla; eighteen ounces of opaque, greenish-yellow, somewhat glutinous fluid evacuated. This was followed by appearance of Skodaic resonance from clavicle to fourth rib; and next day the heart's apex visible in fifth right space close to sternum, but with no other marked alteration in physical signs; nor did the pyrexia abate. On the 19th accordingly, when the cardiac pulsations were visible at epigastrium and close to right of sternum, paracentesis was again performed ; sixteen ounces of puriform fluid being withdrawn. The next day the dulness over left front was found to commence above at the fifth rib ; behind, at the angle of scapula ; vocal fremitus was absent over this area, vocal resonance increased, and some pectoriloquy audible at the base. Moreover, fine crackling râles could be heard in front during inspiration. The percussion note from clavicle to fifth rib was almost tympanitic. There was no cough or expectoration, but continued pyrexia, and on the 26th a small amount of albumen in the urine.

It being evident that the pleural sac still contained pus, and the dulness involving the whole of the left back, except an oval area of resonance in vertebral groove from seventh to twelfth spine, where breath sound was bronchial (it was very feeble elsewhere), the cavity was opened by an incision in the sixth space, mid-axilla, and a portion of the seventh rib resected. A quantity of curdy material was scooped out, and fully three ounces of pus evacuated.

From this date the temperature fell within normal limits, and recovery, though slow, was uninterrupted. The patient left the hospital at the end of July.

The following case is an example of the supervention of the rare and often ominous complication—parotitis. The subject of the attack was a robust young man, and he came under treatment on the first day of his illness, which began very suddenly, and was at first marked by continued vomiting. The rapidity with which the physical signs of consolidation appeared is also worthy of note. He was much prostrated by the illness and convalescence was retarded, not only by the parotid bubo, but by pleuritic effusion. He eventually recovered completely.

CASE XV

Pneumonia of right apex—Pleuritic effusion—Parotitis—Recovery.

Alfred T., æt. nineteen, labourer, admitted into the Middlesex Hospital on March 7, 1889, within six hours of being attacked suddenly with acute pain in the right shoulder-blade, and a fit of shivering. His previous health had been good, and he was steady and temperate.

On admission.—T. 104°. P. 138. R. 40. Face flushed, pain in right side and back, so that a hypodermic injection of morphia was administered. Fifteen minutes later he vomited (he was admitted about 9 P.M.) and continued to vomit at intervals through the night.

Next morning he seemed very prostrate, the lips livid. He was coughing frequently, and the expectoration was tinged with blood. In front the chest was resonant, but a few râles were audible in every part. Behind there was dulness from the apex to the middle of the right scapula, and the breath sound was bronchial. At the right base and over the left lung posteriorly the breathing was weak. The vomiting continued, it was now bilious. The pulse was small and thready.

Free stimulation was prescribed and rectal feeding. An ice-bag was applied to the apex of the right lung, front and back. These relieved the pain, but were with difficulty kept in position.

The vomiting ceased on the 9th. There was now bronchial breathing over the first two interspaces in front (right). Sputum rusty; urine deficient in chlorides.

On the 10th respirations 50. Sputa rusty and copious. Considerable pain on the right side, so that leeches were applied and the ice-bags replaced by poultices. He had become very prostrate and the amount of stimulant was increased. The urine contained a trace of albumen, chlorides still diminished.

In the evening marked friction sound audible over the right infra-mammary region.

On the 11th the right back was dull throughout, and well-marked tubular breathing was to be heard over it. The heart's apex beat was noticed to be just outside the left nipple, so it was inferred that the pleuritis had produced effusion. Tongue dry. Pulse 132. He was slightly delirious during the night. Chlorides almost entirely absent from urine.

A mixture containing ether and ammonia was prescribed.

On the 12th it was noted that the percussion note from the right clavicle to the second space had now become Skodaic in character, there was bronchial breathing here and fine crepitation. From the third rib downwards friction. Bronchial breathing at apex of axilla. Behind there was dulness from apex to base, with tubular breathing, crepitation and friction. At left posterior base impaired resonance and some fine crepitation. Sputum scanty and rusty.

Iced compresses were now applied to the chest instead of poultices, and the quantity of stimulant increased. The apex beat was in the sixth space, half an inch outside nipple.

On the 13th the compresses had to be omitted owing to the delirium and restlessness. Stimulant reduced. The crisis now took place (*seventh* day).

On the 16th the resonance had improved over the right front, but inspiratory crepitations were still present. Posteriorly there was resonance in supraspinous fossa; below this dulness. The breath sound over scapula almost amphoric in quality.

Ammonia and bark ordered, and less stimulant.

On the 17th he complained of pain in the region of the right parotid gland, which was tender. It was painful to move the jaw. The apex beat was now in normal position. Pleuritic friction audible over right side.

On the 18th the swelling, pain and tenderness in parotid region had increased. Hot fomentations applied.

On the 20th swelling increased, redness, but no fluctuation.

On the 21st it is noted that the swelling extends from mastoid process to angle of mouth; the skin over it is dark red, and œdematous. Temperature 101°. Physical signs clearing up.

On the 23rd an incision was made into the swelling and a small quantity of thick pus escaped; this gave relief. The breathing was becoming more vesicular over right back.

On the 25th there was some discharge from the right auditory meatus; a drainage tube had been inserted into the abscess and the swelling was fast dwindling.

On the 31st it is stated that the temperature was still ranging between 100° and 101°, and that there was some inflammatory thickening and induration over parotid. The fomentations were continued and mercurial ointment applied. Physical examination showed that there was dulness behind from angle of right scapula to base; in front from base to fourth rib in nipple line, and to fourth rib in mid-axilla, where he still had pain. Breath sounds weak and distant, no friction. A hypodermic syringe was introduced into the chest in axilla, but no fluid found.

April 6.—Temperature still varying between 100° and 101°. The right side moves but little, and signs still indicative of pleural effusion in right side, the breathing being almost inaudible from angle of scapula downwards.

On the 18th it is stated that he does not gain flesh and that he sweats much at night.

He gradually improved from this date, and left for Eastbourne Convalescent Home on May 24.

Appended is the record of the pulse and respiration rates and the temperature for the first month of the patient's stay in the hospital. The temperature was taken every four hours, but a sufficient idea of its general course is gathered by utilising only the record for 6 A.M., 2 P.M. and 10 P.M. The observations on pulse and respiration taken at 10 A.M. have now, for convenience, been transferred to the 6 A.M. record.

Date	6 A.M.			2 P.M.			10 P.M.			—
	T.	P.	R.	T.	P.	R.	T.	P.	R.	
Mar. 7	—	—	—	—	—	—	102	—	—	
,, 8	103·4	128	40	103·6	—	—	102	120	40	
,, 9	100·6	108	40	101·8	—	—	101	128	32	
,, 10	101	—	—	102·6	—	—	102·8	—	—	
,, 11	102·6	132	52	102·4	—	—	100·2	—	40	
,, 12	102·2	140	52	103·6	—	—	102	—	—	Iced compress applied
,, 13	102·2	—	—	103	—	—	96·8	108	44	Crisis
,, 14	96·4	114	36	99·8	—	—	100·2	108	32	
,, 15	99·4	96	30	98·4	—	—	98·4	—	—	
,, 16	97·4	120	32	99·2	96	32	98·2	—	—	
,, 17	98	98	28	98·2	—	—	99·2	—	—	Parotid swelling
., 18	100·4	96	28	100	—	—	100·6	100	28	
,, 19	99·6	108	—	101·2	—	—	99·4	—	—	
,, 20	99	—	—	100·2	—	—	99	—	—	
,, 21	99·8	108	28	101	—	—	100	—	—	
,, 22	98·4	90	32	101·4	—	—	99·6	—	—	
,, 23	98·2	90	20	98·8	—	—	100·2	—	—	Incision into abscess
,, 24	98·4	88	24	99·8	—	—	100	—	—	
,, 25	98·4	—	—	99·6	—	—	100·8	—	—	
,, 26	99·4	96	24	102·2	—	—	101·6	—	—	
,, 27	101·4	–	—	99·4	—	—	102·2	—	—	
,, 28	99·4	—	—	100·6	—	—	101·4	—	—	
,, 29	99·4	96	28	99·6	—	—	100·8	—	—	
,, 30	99·4	114	28	100·4	—	—	101·8	—	—	
,, 31	99·2	96	32	100	—	—	100·8	—	—	
Apr. 1	99·2	108	26	100·4	—	—	99·4	—	—	
,, 2	99·2	102	32	101·6	—	—	101·2	—	—	
,, 3	98·8	—	—	99	—	—	100·8	—	—	
,, 4	98·8	102	36	98	—	—	101·2	—	—	
,, 5	98·6	—	—	98·4	—	—	101·8	—	—	
,, 6	99·6	—	—	99	—	—	100·6	—	—	
,, 7	98·6	—	—	100·2	—	—	102·2	—	—	

The evening temperature did not become normal until May 1.

Case XVI

Pneumonia—Meningitis.

Thomas W., æt. twenty-five, a van washer, admitted into the Middlesex Hospital, under the care of Dr. Cayley, on March 25, 1889. He had had syphilis, and seven years before fractured ribs on both sides. He used to drink heavily, but had latterly been more temperate. His occupation exposes him much to the weather, and two years ago he is said to have had an attack of pneumonia.

On the morning of March 24 he had an attack of shivering and feverishness, with headache, and commenced to cough, some dyspnœa, and twice vomited.

He was a strongly-built man, but on admission was very prostrate, breathing rapidly, complaining of pain in the left side, and having a short, hacking cough with scanty, viscid, rusty expectoration. Cheeks flushed, lips dry and covered with sordes; tongue dry and brown. P. 125, small and soft. T. 102°. R. 40. Heart sounds weak but normal; chest capacious. Rhonchal fremitus over both fronts; vocal fremitus diminished over left base. Absolute dulness on percussion from angle of left scapula downwards; and hyper-resonance in front from apex to fourth rib. Normal percussion note over whole of right lung. Breath sounds at left apex were harsh, accompanied by rhonchi. At the base in front and in axilla fine crepitant râles with inspiration; over the dull area tubular breathing, and some inspiratory crepitation. Harsh breathing over right lung with scattered rhonchi, and some crepitation at the base.

The patient was drowsy, but became delirious during the night. On the 26th the delirium continued. He also had frequent vomiting. The expectoration was rusty; cough slight.

The temperature remained at about 102°.

He continued to be drowsy throughout the 28th, but in the evening became actively and violently delirious, followed by great exhaustion, from which he never thoroughly rallied, but died in the early morning of the 29th.

Post-mortem Examination, made by Mr. Leopold Hudson, revealed complete consolidation of the left lung in a state of red hepatisation; there was also marked injection of the bronchi in the right lung. The spleen was soft and deeply engorged; the liver in a state of cloudy swelling. Over the left half of the cerebellum the meninges were coated with puriform exudation. There was no tubercle.

The case illustrates a fact, which has been pointed out before, that intercurrent meningitis in pneumonia is liable to be masked by the signs attributable to the primary disease. In this particular case the habits of the patient might have suggested an alcoholic cause for the violent delirium. The tendency to coma was a notable feature in the case. The limitation of the meningeal inflammation to the region of the cerebellum explains the absence of any definite diagnostic signs of its presence. (See p. 119.)

PART II

VARIETIES AND COUNTERFEITS

CHAPTER X

VARIETIES

Modifications due to the seat and extension of the lung inflammation—to privation of food—alcoholism—pythogenic source—the puerperal state. Recurrent pneumonia—Pneumonia with heart disease.

WHILE pneumonia, as we have shown, is a distinct and definite disease, it will not be supposed that any single description of it can include all its possible varieties. As with other affections, certain symptoms may be here or there unusually prominent, others masked or variously modified. It needs only a clear conception of what constitutes the fundamental character of the disease in order to discriminate easily between its occasional and essential phenomena. It is less difficult, indeed, to enumerate all its observed varieties than to make good the claim of any one of these to be regarded in the light of a type or pattern from which the rest are declensions.

Yet there are certain forms of the disease which, whether for their danger, rareness, or obscurity, demand separate notice. Thus, for example, pneumonia will sometimes spread from its first place like erysipelas. Day by day, from its commencement until crisis, the area of lung involved obviously widens. It may thus extend from one side to the other, as from base to base, or from the base of one lung to the apex of the other (very rarely, as it happens, occupying both apices).[1] Such cases have a special danger: they show early prostration, and sometimes active delirium. Upper-lobe pneumonia is apt to be of this sort, the inflammation starting from a central portion of lung and not becoming obvious to auscultation until the apex is affected. In such instances the symptoms are modified, not because the apex is the part concerned, but because the inflammation spreads, and is not recognised until that part is reached. It is to this so-called erysipelatous form of the disease rather than to the accident of locality that the exceptional degree of prostration is due. It must be admitted at the same time that pneumonia of the apex, even when not of this form, is more often associated with delirium than when the seat of inflammation is lower down. Yet it would certainly be untrue to say on the whole that the prognosis of apex pneumonia is less favourable than that of basic. The contrary is probably the case.

It is to this form of the disease more particularly that the expression 'cerebral pneumonia' has been applied, a term of doubtful propriety, meaning no more than that nervous disturbance is unusually prominent and sometimes of such early occurrence as to precede any physical sign on the part of the lung. Severe headache, active and even violent delirium, convulsion, muscular spasm, and other symptoms hard to distinguish from meningitis, may usher in a pneumonia which in its later stages presents no peculiarity whatever, and quickly recovers. We have already, recorded (Chapter IX) the case of a young woman who exhibited maniacal delirium as a prelude to pneumonia of the right apex of slight extent and not severe. In another instance, that of a boy of eight, admitted into the Westminster Hospital, an epileptiform fit occurring a few hours after the appearance of pyrexia, was followed in three days by consolidation of the left apex, the patient recovering by crisis in the usual way.[1] Nervous symptoms of this sort are commonest in early life, and will be mentioned again in connection with the pneumonia of children.

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[1] In regard to the relative frequency of pneumonic inflammation in the several parts of the lung respectively, the following may be quoted from the 'Collective Investigation Report' (page 45), where the exact figures for 1,048 patients are shown in tabular form.

The right lung is more often affected than the left, the left more often than the two together, the numbers respectively being 455, 327 and 266. The most common seat is the right base (which is the part affected in more than a quarter of the whole number). By far the rarest seat is at the two apices. Only 4 such cases occur in 1,048. Next in frequency to the right base is the left base, and next again both bases. Then, with considerable interval, come in descending order the whole of the right lung, the right apex, the whole of the left lung, the left apex. The proportion of deaths for the several localities can only be estimated very generally and for the higher numbers. The death rate is highest where both lungs are involved, being more than double of that where only one side is involved. There is no appreciable difference of mortality between the two sides. The subject of local seat in reference to mortality has been discussed under Prognosis (Chap. VIII). See also the analysis given in Chap. V, p. 87.

Not seldom the recognition of pneumonia is made difficult owing to the previous habits and mode of life of the patient, as when it attacks those *addicted to alcoholic excess or weakened by want of food and other privations that attend extreme poverty.* The combination with delirium tremens will be best considered in the next chapter, along with other acute diseases which modify the ordinary course of pneumonia. Starvation and alcoholic poisoning have much in common in their relation to this disease. Both predispose to a form of pneumonia whose advent and progress are habitually so insidious that only physical examination can detect it. Thus an individual may exhibit signs of prostration which seem sufficiently explained by poverty and starvation, or which suggest enteric or typhus fever. By-and-by, and often quite suddenly, he will die, not of fever or mere starvation, but, as is shewn after death, of pneumonia. Such an issue takes us by surprise. Yet, inasmuch as these patients exhibit no open lung symptoms throughout, it argues no great want of vigilance that the nature of the affection should have been overlooked in lifetime. It may be so rapid that a careful exploration of the chest shortly before death fails to discover any sign of lung consolidation; and so stealthy that the patient sometimes remains at his work almost to the time of death, thus seeming to exclude the supposition that an acute pneumonia has run its course to a fatal issue by the usual stages.

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[1] See 'Lancet,' March 9, 1878. Dr. Farquharson in a paper on the 'Forms of Pneumonia,' quotes the case of a youth of eighteen where muscular rigidity, passing into unconsciousness, preceded the first notice of inflammation at the base of the right lung.

It is beyond question that cases of this sort have from the first a character of their own. With extreme prostration the local affection may be of limited extent, temperature not greatly raised and the pulse not frequent. Trousseau records an instance where, with a respiration of 88, the pulse was but 84.[1] These are not examples of a benign disease which, with good care, tends to recovery, the definition which applies usually to pneumonia. They exhibit from the first a destructive energy beyond the reach of remedies, and of which death is the natural and inevitable end. Yet, while they are so far distinct, it is difficult to exclude them from the category of pneumonia unless we determine to limit that disease by other restrictions than those that have hitherto served.

The best illustrations of this form of disease occur in middle or advanced life, away from hospitals and where death is sudden and unexplained, so as to render a coroner's inquest necessary. It is from this source, indeed, that Dr. Robert Lee kindly supplied to the first edition of this book some notes illustrative both of the suddenness of death, the social condition of the patients, and character of the local inflammation. The following may be here quoted :

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[1] Trousseau, vol. iii. p. 358; 'Clin. Med' New Syd. Soc.

CASE 1.—An inquest was held before Mr. Bedford on the body of a woman, fifty-five years of age, of dissipated habits, who had been found dead early in the morning in her bedroom. She had come home the previous evening seeming as well as usual, and had retired to bed without complaint. She was kneeling at the bedside when her room was entered in the morning, and had been dead but a very short time. The only disease that was discovered on examination was universal grey hepatisation of the lower lobe of the left lung ; all the other organs were healthy.

CASE 2.—A respectable man, aged forty-eight, was found dead in his bed, and an inquest was necessary. For three years he had worn a tube in the trachea in consequence of laryngeal disease, for which he had undergone tracheotomy. He had been unwell for two days previous to his death, but not so seriously as to excite any apprehension. The upper lobe of the left lung was entirely solid from grey hepatisation. The disease was limited to this part, and there was no evidence of inflammatory changes in other parts of the lungs. All the other organs of the body were healthy.

CASE 3.—An examination was made of the body of a man who had disappeared, without any reason, about ten days before, and who, it was proved by the evidence adduced at the inquest, had committed suicide by drowning in the Thames. He was of middle age, and had been attending at Guy's Hospital as an out-patient for a few days before his death. On the day his death occurred he had been driven to the hospital, and had afterwards left his sister's house late in the afternoon with the intention of walking home. Nothing was heard of him again till his body was found in the river. The whole of the right lung was in a state of grey pneumonia. The left lung and the bronchial tubes were quite free from any of the usual appearances of acute inflammation. The other organs were healthy. It was suggested that the act of suicide was committed in a state of mental derangement, a not unfrequent symptom of pneumonia.

In such cases the poverty and neglect are necessary factors, and often preclude the possibility of early observation on the part of the physician. Illness is concealed if not unfelt ; and the fatal event seems so sudden as to suggest that the grey stage of hepatisation is reached at once, a view held by Dr. Hodgkin;[1] yet it would seem more probable that the successive stages of pneumonia are very rapidly fulfilled owing to the lowered vitality of the subject. The condition revealed after death is essentially pneumonia, differing in nothing from that of a patient who, by the usual steps, has reached the same stage. Anatomically, that is to say, the inflammation is unilateral and sometimes of the upper lobe, it is strictly limited; there is little accompanying bronchitis, the pleura is implicated, while the heart, kidneys, and other organs are often healthy.

Distinct from these (though the distinction may not always be suffered to appear, owing to an imperfect history, or partial and inexact post-mortem inspection) are certain examples of consecutive pneumonia where the disease is but the final stage of a prolonged illness, of which the general character and consequences may be gathered from inspection of other parts of the body: dwindled, granular kidneys with diminished cortex, a thin and dilated right ventricle, often encroached upon or changed by fat: not in one but throughout both lungs evidence of bronchitis; while the organ chiefly affected, though solid, is yet compressible, and, carefully looked at, is seen to be consolidated by lobules or collections of lobules, exhibiting, between these irregular tracts of mere congestion, a condition which it shares with the other lung. Both are, indeed, similarly affected, though in different degrees, insomuch that such cases are often described, living and dead, as 'double pneumonia.' There is seldom any fibrinous pleurisy, but often one or both pleural cavities contain fluid. The subject of this condition has suffered many previous attacks similar to that which at last kills him. We shall endeavour to show in its place that the points of resemblance to pneumonia are here apparent and not real, and that the old physicians were right in speaking of it as false or bastard pneumonia (Chapter XIV).

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[1] 'Lectures on Pathology,' p. 98.

At the same time true pneumonia, as we have shown, may occur with renal disease both acute and chronic, and with the latter is wont to assume a latent character very similar to that poverty form of which we have just given illustrations (see Chapter VI, p. 115).

There is a form of pneumonia now commonly known as *pythogenic*[1] allied to enteric fever, and occurring in similar circumstances from sewer gas, imperfect drainage, and like causes. The proper place for its discussion will occur under Etiology; but, in the meanwhile, something may be said here as to its clinical characters.

In some cases of this origin, at all events, the common pattern of pneumonia is not preserved. The patients manifest symptoms of septic poisoning, with acute pulmonary congestion, and sometimes lobular consolidation. There is no pleuritic stitch, perhaps no expectoration, and in some instances, diarrhœa with loose, ochre-coloured stools. The lung affection is often double, and its physical signs, even at an advanced period of illness, may amount to no more than crepitation of medium size. When these patients die, death is not so much from the local as from the general affection, and the manner of sinking is strikingly suggestive of enteric fever. On post-mortem inspection, too, the lungs are not hepatised, but solid, or perhaps rather carnified, in patches. There is, moreover, no recent lymph on the pleura and no pericarditis.

The subjects of this form of disease are often those who have been exposed to the very same influences that have sufficed at the same time to induce enteric fever in others of the same household. The lung condition, indeed, nearly resembles that which arises in actual connection with typhoid, and is no doubt sometimes confounded with it.

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[1] See Drs. Grimshaw and Moore, Pythogenic Pneumonia, 'Dublin Journal of Medical Science,' May 1874.

But, on the other hand, a pneumonia of pythogenic origin may be indistinguishable from that due to chill or exposure, except, perhaps, for the greater prominence of gastric symptoms. The following is one amongst many illustrations that might be given of pneumonia of this source.[1]

Lizzie C., twenty-three, married, was admitted into the Middlesex Hospital on January 29, 1885, suffering from pneumonia. She had been attacked the day before in the morning with giddiness, so that she had to leave off work, and during the rest of the day she had shivered frequently, vomited several times and suffered from headache and pyrexial pains. It appeared that on the 22nd and on the 27th she had been charing at a house in Bloomsbury Square, where one of the inmates had been ill for some months, but that on the morning of the 28th, a few hours before she felt ill, she was engaged at work in a room in N—— Street, where there was a noxious smell.

On admission her temperature was 104·4, pulse 120, respirations 24. The cheeks were flushed, lips dry and covered with sordes; *alæ nasi* working. She complained of headache and malaise, but of no pain in the chest. (There had not been any stitch.) The chest expanded well and equally and the vocal fremitus was equal. Nor was there any dulness on percussion in any part. On auscultation, however, there was some whiffing breathing at the angle of the right scapula, and a few fine crepitations in the same region. The heart sounds were weak, and at pulmonary cartilage a systolic bruit was audible. There was also a venous hum in the neck. Abdomen natural; urine 1012, acid, no albumen. During the day and following night the temperature remained high (103° to 104°). Next morning, P. 144, R. 20; headache very severe. Some impaired resonance in right interscapular region, and breath sounds more bronchial at angle of scapula. The temperature fell during the day to 100°, pulse to 104, and resp. to 20.

January 31.—T. 103·8, P. 116, R. 22. Very sick in night, had no sleep; headache still bad. The bowels had been moved by a dose of calomel, motions pale and typhoidal in appearance. Has a slight cough, sputa scanty, mucoid, not rusty. The physical signs had not become more definite, only a few crepitant râles being heard at right apex. On this day at 2 P.M., and again at 6 P.M., when the temperature was respectively 103·6 and 104·6, the patient was immersed in a bath at 85° for ten minutes. She bore this well and seemed the better for it, the temperature after the second bath remaining down to 102° or 102·6 until 6 o'clock the next morning.

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[1] Examples of pneumonia occurring in groups, and attributable beyond reasonable doubt to a pythogenic origin, will be quoted under Etiology, Chap. XVI.

February 1.—T. 103·6, P. 116, R. 44. Still has a hacking cough, but no further development of physical signs. No expectoration.

February 2.—Headache less. The pyrexia much lower; at 6 P.M. T. only 100·4.

February 3.—T. 102·4 to 100·6. Slight bronchial breathing, with bronchophony at right apex, and a few crepitations.

February 4 (*eighth day*).—Sudden fall of temperature to 97° and of pulse to 56. Slight dulness at right posterior apex, and breathing rather blowing. Scattered râles at base. In anterior apex crepitation, but no bronchial breathing. Sputa muco-purulent, containing specks of black pigment. There was no further rise of temperature, and the slight signs of pneumonic consolidation rapidly cleared up. She left on the 19th.

If this attack were really attributable to the foul air of the room where the patient was working on the morning of January 28, the signs of illness rapidly ensued on exposure to the miasm. The most notable feature of the case was the high pyrexia and the very slight physical signs, or indeed symptoms of lung involvement. But it was obviously a case of pneumonia, in which the pulmonary lesion was very limited. There was a definite crisis on the eighth day, yet at no time was there characteristic expectoration, or any pleuritic signs.

Here may be a fitting place to notice the occurrence of *pneumonia in the course of pregnancy.* Observations upon this subject are not sufficiently numerous or detailed to warrant the expression of any confident opinion. But there exists a general belief among obstetric physicians, which is shared by Grisolle, that acute idiopathic pneumonia (septic conditions being excluded) has a special danger for puerperal women, and that the peril is greater before rather than after the seventh month.

Of all acute diseases it is one of the most likely to produce abortion, while, contrary to the rule prevailing in variola, this event is regarded as of favourable augury. An enormous rate of mortality has been ascribed to pneumonia thus occurring, but the evidence is scanty,[1] and, except for its fatality, we are not informed that pregnancy modifies the symptoms or the course of the disease. The silence of most authors on the subject, and our own experience, points to the conclusion that this untoward concurrence is rare.

In the following illustration pneumonia affected the right apex, crisis occurred on the eighth day, and abortion of a six months' fœtus followed thereupon. Though the symptoms were severe, the woman made a good recovery.

Sabina C., twenty-six, cook, unmarried, one child, no miscarriage, healthy-looking and well-nourished, was admitted to the Westminster Hospital March 25, 1888. She was quite well until a sudden attack of shivering three days before admission; she went to bed at once and shivered all night. No previous illness known, except scarlet fever when about twelve; she is of temperate habits and healthy family history.

On admission she lay low in bed; had extreme dyspnœa, the face distressed, flushed and dusky; some herpes on upper lip; R. 88, P. 140, T. 103·6. Sputum typically rusty; tongue shiny, dryish, and glazed; urine acid, of normal specific gravity, containing a trace of albumen. Physical signs of consolidation with marked dulness and tubular breathing at right apex. Some general bronchitis.

On the fifth day the patient was breathing with great difficulty, the face distinctly cyanosed; there was slight diarrhœa. P. 152, R. 52, T. 104°; sputum still rusty.

On the seventh day the temperature at 8 P.M. reached its highest point, 104·6; pulse was over 150, and the distress from dyspnœa very great. The face flushed and still slightly cyanosed; herpes still well displayed; the woman very prostrate.

From that highest point and from that time temperature began to descend gradually and without interruption. It thus continued in descent all through the eighth day and until 6 A.M. of the ninth day, when it registered 97·6 (on a two-hours temperature observation). During this, while there was little abatement in the other symptoms, pulse was 160, respirations 56, and dyspnœa acutely distressing to the patient. At 9 A.M. of this same ninth day the temperature stood still (having risen to the normal), and the woman aborted; the fœtus, in the opinion of the obstetric physician, being of about six months.

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[1] See Cazeaux's Midwifery, 'Bullock's Trans.' p. 359.

Normal temperature was maintained throughout that day; and on the following (tenth) day pulse had fallen to 104 and respirations to 48. The patient continued very weak for a fortnight longer, and the consolidated lung very slowly resolved, but there was no return of pyrexia, and a fortnight later she was practically convalescent. Days in hospital, 38.

The case is a fair sample of its kind, being free from complication and occurring in a previously healthy person. It is remarkable from the extreme distress caused by dyspnœa and the marked cyanosis, the amount of lung involved not being considerable. Such symptoms are probably attributable directly to the pregnancy, and go to justify the opinions just quoted as to the gravity of pneumonia in puerperal women. It appears, at the same time, that the duration of the disease, its behaviour as a local inflammation, and the character of crisis are not altered by this conjunction. The occurrence of abortion precisely at the end of crisis is also remarkable, its sole consequence being, so far as was seen, to put back by some fortnight the usually prompt recovery that characterises pneumonia.

Relapsing pneumonia is so rare an event that it may seem hardly to require separate consideration. Grisolle estimates the proportion of relapses at about one-twenty-eighth of the whole number of cases; while Briquet puts it at one-fifth. Such wide discrepancy probably depends upon laxity of definition. Thus, broncho-pneumonia, in which relapses are common, may be mistaken for lobar, and even in the latter a brief remission of pyrexia may concur with such general amendment as gives hope of impending crisis, which the next day disappoints. By relapsing pneumonia, however, we mean more than this, namely, the complete arrest or suspension of pyrexia for some days, with resolution more or less complete, followed by reappearance of the early symptoms and repetition of the inflammatory process ; we mean such recurrence, that is to say, once or twice or thrice, of the morbid phenomena as we encounter not seldom in enteric fever ; and this is certainly very rare in lobar pneumonia.

It is true that in the spreading form of the disease the pulmonary inflammation sometimes makes pause. Both temperature and pulse moderate, and some phenomenon of crisis, such as sweating, strengthens the belief that the disease has reached its term. But in a day or two, with a fresh rise of temperature, a fresh area of lung will become inflamed, and the patient undergoes a second attack similar to the first. Yet it must be pointed out, first, that in spreading pneumonia the pyrexia does not always behave in this way, but that more often, after a continued high temperature of six or seven days, a sudden crisis brings the extending inflammation to an end in the manner characteristic of the disease in its ordinary shape ; and, secondly, that if the term relapsing or intermittent pneumonia may be applied to these brief remissions, it is not to be understood in the same sense as in enteric fever, where the whole array of symptoms from first to last may be exactly repeated, once or twice or thrice, or even more often.

The following narrative shows how a false crisis sometimes interrupts the course of pneumonia, and divides it into two parts.

Alfred Dickinson, aged 28, indoor servant, well-nourished and of previous good health but for slight cough, was admitted to the Westminster Hospital April 4, 1889. Three days before admission, while at work, he shivered and had sharp pain in the left side, increased by coughing. The following day he spat up some blood-coloured sputum, complained of headache and sickness, and had to leave work. On admission decubitus is dorsal, cheeks flushed, herpes on lips, skin hot and dry, T. 103°, P. 120, and R. 44–50. Signs of consolidation were distinct and well marked as regards the lower two-thirds of left lung, vocal fremitus and voice ring being increased, together with tubular breathing and dull percussion note over that area.

On the fifth day of illness respirations had risen to 60, evening temperature was 103·6, pulse somewhat less frequent, general condition unchanged. On the sixth day, however, *temperature fell to* 100°, *and continuing in descent* (save for a brief evening rise to 102°) *reached* 99° *on the seventh day, when the pulse was* 96, *respirations* 42, *and the patient had profuse sweating and felt better.* The same evening, however, temperature again rose irregularly, and on the ninth day reached 102·6, pulse 120. And now crepitant rhonchus, with some impaired resonance, was audible in a fresh place, namely, at the apex of the *right* lung. Along with this second pyrexia, which lasted four days, signs of resolution (redux crepitation, &c.) were audible in the lung first attacked, shortly followed by similar signs on the part of the other. Convalescence was delayed in this case by a small empyema of the left side, which was aspirated and did not return, but what was in fact the second crisis occurred on the eleventh day of illness ; a week later the man had gained four pounds in weight and was soon after convalescent.

In a case we have related elsewhere,[1] a weak anæmic girl of eighteen two days after crisis and when convalescent of a pneumonia affecting the left lung, suffered relapse and underwent a second period of pyrexia and prostration worse than the first. Physically this event was signified by the fact that a portion of the lung consolidation of the first attack did not share in the recovery of the rest, but remained unresolved until the second defervescence occurred.

In the following example three distinct pyrexial periods followed the first attack, temperature being subnormal in the intervals, the patient dying on the 28th day.

John Murphy, aged 20, seller of newspapers, a healthy-looking youth, but described by his uncle as of irregular and rather intemperate habits, was admitted to the Westminster Hospital, January 5, 1890, suffering from pains about chest, limbs and back, headache and like symptoms of three days' duration, commencing with rigor, his illness at the first suggesting the then prevalent influenza. On admission his temperature was 102°, rising to 103° the same day at noon, and in steady ascent, upon a two-hour chart, during the next (or fourth) day till it reached 104·8, pulse being only 104. Not until then were the first physical signs of pneumonia discovered in some crackling about left axillary region, and tubular breathing at angle of left scapula. The patient was delirious that night and during the following day, but the temperature tended downwards, the pulse being 110 and irregular; respirations laboured but not frequent, 38 and thereabouts, except in paroxysms, when they exceeded 50, the pulse also rising the while from its moderate rate just mentioned to over 120. Such was the condition on the fifth day.

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[1] 'Lancet,' March 22, 1880. Dr. J. T. Collins describes pneumonia of left lung in a child of eight, where two days after crisis the right lung was attacked more severely than the left, 'two attacks, one for each lung, each lasting five days, with a crisis between.' 'C. I. C. Report,' vol. ii Case 780.

From this man's age and appearance and the general character of the symptoms, a confident prognosis would have been given of ultimate and probably critical recovery, notwithstanding the exceptional prominence of delirium. But there were three elements of doubt : the possible commencement in influenza, a dangerous antecedent of pneumonia as had been amply shown by the epidemic of 1889-90 ; the alleged intemperate habits ; and more than all the history of an attack of acute nephritis with œdema two years before, and the persistence of albumen in large amount. Resuming the narrative :

On the evening of the fifth day temperature began to descend, slowly but continuously, so that from 104° it had descended to 102·4 by the early morning of the sixth day; but there was no general improvement or abatement of delirium, and on the seventh day (temperature still in descent) he had a paroxysm of dyspnœa which much exhausted him, respiration exceeding 50 and pulse 120. The area of inflammation had also increased upwards. Thus, while *the chart-tracing* (taken every two hours throughout the illness) *indicated a defervescence by lysis, and temperature on the eighth day was but* 99°, his state was obviously deteriorating.

On the following (or ninth) day the *right* lung, exempt until then, showed signs of consolidation of the lower lobe. On the same day *temperature rose abruptly from* 99° *to* 104°, whence it immediately began to descend during the tenth and eleventh days, and, slowly falling, had reached 95° on the early morning of the twelfth day. Diarrhœa had now set in; there was extreme prostration, with drying tongue, quiet delirium, and extreme aversion to food, as well as to alcohol. Pulse had fallen to below 100, and respirations, laboured and difficult, counted only 28. There now occurred an interval of four days during which the patient on the whole somewhat improved, being less prostrate, with little delirium and obtaining sleep; but he vomited after food several times, and his tongue was brown coated, dry, and 'baked,' and with difficulty protruded. (For some days past large, flat, indolent pustules, with broad red areolæ, have been noticed over buttocks and lower part of back, the older of these are tending to dry up; there is no sloughing or active suppuration.)

On the 17th day he had *a second temperature rise*, with quickening pulse and respiration 120 and 30 respectively. This lasted two days, and was followed, after an interval of four days, by *a third and more marked recurrence of pyrexia*, *temperature exceeding* 103° *for three days* (that is until death), while the pulse now, for the first time, reached, and presently exceeded, 130 (130–140).

It could not be certainly said from the physical signs that the local inflammation continued to spread in this later stage as it had obviously done in the earlier. The part of the *left* lung originally attacked had now recovered, and such tubular respiration as remained on the right side was perhaps audible somewhat higher up, as though consolidation had extended on this side, as, in the earlier stage, it had certainly done on the other. On the day following this observation (26th of illness) the patient had a fit of general convulsion, and two days later he died. The condition of the urine varied slightly from day to day as regards albumen, it never indicated a trace of blood, and was never observed to be scanty, although the existence of diarrhœa and delirium made continuous examination impossible.

Post-mortem.—The body was emaciated. Over sacrum and buttocks are numerous small, ragged openings leading into pustules or small abscesses of the subcutaneous tissue.

With the exception of the middle lobe and a small area next the diaphragm, the whole of the right lung is in a condition of grey hepatisation. The pleural surfaces are united over upper half by recent lymph. Below this there is lymph and fluid. The left lung is œdematous and its lower lobe firmer than natural, but it is quite crepitant and floats in water. There was some soft, not recent adhesion of left pleura.

Heart's left ventricle is not hypertrophied. There is much firm, discolorised clot in the cavities.

The kidneys (3¼ and 4½ oz.) are large, pale, and mottled; their capsules free; the cortex increased and mottled with small irregular areas of reddish purple; the pyramids very pale. There was nothing special to observe of the other organs.

This youth, presenting at first none but the ordinary signs of pneumonia, from which one of his age and appearance would almost certainly recover, was rendered, as has been said, an unsuitable subject for the disease, owing to a distinct history of acute nephritis two years before, and to habits of intemperance, as to which the evidence was less precise. After his temperature had declined by lysis on the 7th day he underwent three distinct and separate pyrexial periods, in the intervals of which the temperature was subnormal. These several periods were signified with some precision (especially the first, where the pneumonia leaving the left lung attacked the right) by physical signs indicating an extension of the area of inflammation. But the periods of pyrexia and the intervening periods of subnormal temperature made no difference as to the patient's general state, which, with some short breaks, was one of continual decline, and, strictly speaking, the case is not one of recurrent pneumonia, but of recurrent pyrexia.[1]

It was remarkable that the left lung, which was first attacked, recovered, while the right, suffering later, exhibited grey hepatisation. It was apparent, both post-mortem and from clinical observations from day to day, that the inflammation, though spreading from side to side, quitted the left lung at about the same time that it invaded the right, where its progress was continuous up to the time of death. Thus we had, in the same subject, a recovering or recovered lung along with one showing grey hepatisation, the difference in the conduct of the exudation on the two sides being determined, as it would seem, by the fact that the first inflammation concurred with a fuller power of resistance on the part of the patient than the second.

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[1] A striking example of recurrent pneumonia, occurring in a man aged 82, is related by Sir Andrew Clark. In the author's words this patient 'in the course of a seven weeks' illness, after having passed through nine or ten severe rigors, and six small successive attacks of pneumonic exudation, was declared to be quite well.' The case in Sir Andrew Clark's opinion is 'rare, if not unique in the annals of medicine.' 'British Medical Journal,' December 20, 1884.

We have some hesitation in including among the varieties of pneumonia certain cases forming a group by themselves in *connection with disease of the heart.* The so-called cardiac lung will be described elsewhere among other conditions distinguishable from pneumonia (Chapter XIV). But it is beyond question that there is a form of this latter affection to which valve disease, especially mitral stenosis, adherent pericardium, and fatty degeneration in their several degrees, contribute. Here, in addition to pulmonary infarcts, hæmorrhagic extravasation, and engorgement, familiar consequences of cardiac lesion, we find besides hepatisation and recent pleurisy, confined usually to one lung. The most characteristic examples are those of young persons with extreme mitral narrowing, the result of past rheumatism, together with general anasarca.

This form of hepatisation may be without pleurisy, and it may continue long in one stage. It has little tendency to undergo the later stages of grey infiltration, and a fair proportion of patients recover. It thus differs strikingly from cases associated with chronic kidney disease. Sometimes the well-marked character of the physical signs attracts notice, but perhaps more often general œdema and cardiac distress divert attention, and in the absence of the more obvious symptoms of lung inflammation the disease is only disclosed after death. No doubt this form of pneumonia is rare, yet it is sufficiently distinctive in both clinical and anatomical features to deserve notice.

Out of seventeen cases of extreme mitral stenosis,[1] exhibiting the characteristic lung changes of infarcts and scattered hæmorrhages, there are four (or five) with pneumonia as well. Of these the following may be mentioned.

A woman of twenty-eight, with physical signs of mitral stenosis, but who had never had rheumatism so far as was known, was admitted with general anasarca and (for a few days only) had bloody expectoration. Dropsy increased, and without marked pyrexia or dyspnœa she slowly died.

Post-mortem.—The whole of the right lung was solid, the left in a similar condition less extensive and less advanced. There was no recent pleurisy. The mitral orifice was much contracted and thickened by means of calcareous deposit, only the little finger could be inserted. The kidneys were increased in size and the cortex swollen ; other organs healthy.

In the following case mitral stenosis (the result of bygone rheumatism) probably determined the fatality of pneumonia occurring in the course of acute rheumatism, while, at the same ime, it predisposed the patient to the lung inflammation.

A boy of seventeen was admitted with acute rheumatism of four days' duration, and had physical signs of left pneumonia. He died in six days, or ten from the commencement of rheumatism.

Post-mortem.—The whole of the lower and part of the upper lobe of the left lung was in a state of red hepatisation, the pleura unaffected. The right lung was also partly consolidated. There was extreme mitral stenosis, the orifice barely admitting point of forefinger. The other organs were healthy.

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[1] In the first edition of this work these cases were printed in tabular form. They refer to the practice of St. George's Hospital at the time one of us was medical registrar at that institution, and were originally inserted by permission of its medical staff.

A man of fifty, who had suffered cough and dyspnœa for many years and had valve disease, probably of rheumatic origin, was on admission cyanosed and much distressed from dyspnœa, but without pyrexia. On admission sharp râles were heard at the base of the right lung, but there was no evidence of consolidation. Without undergoing notable change he shortly died.

Post-mortem.—The lower lobe of the right lung was found hepatised and covered with recent lymph. There was old pericarditis, the muscular structure of the heart was fatty, the mitral orifice narrowed, admitting only one finger. The surface of the kidneys was slightly granular. There was no other disease.

CHAPTER XI

PNEUMONIA ASSOCIATED WITH OTHER ACUTE DISEASES

Acute rheumatism—Pericarditis—Delirium tremens—Acute nephritis—The specific fevers—Diphtheria.

Apart from complications, which have been already considered (Chapter VI), certain acute affections are apt to concur with pneumonia in such manner as to mask or modify its symptoms and make diagnosis difficult. The nature and intimacy of this alliance will be seen to vary with particular examples of it, while the conclusions thence derived concern the pathology of the disease, and will be discussed hereafter. We have now to examine the claims of certain affections respectively to be regarded as allied to pneumonia, on the ground of their more or less frequent concurrence with it.

When enumerating the many forms of pneumonia recognised by authors we mentioned the rheumatic. Understanding this term as applying to pneumonia arising in the course of *acute articular rheumatism*, it may perhaps be doubted by some whether the event is sufficiently common, or the pulmonary inflammation, when so related, sufficiently special to justify the admission of such a variety. Whatever we might be led to expect from a consideration of the pathology, subjects, and exciting causes of the two affections and their meeting-point, so to speak, in pericarditis, yet in the guidance of clinical experience it must be admitted that, although both diseases are common at about the same age, the occurrence of pneumonia in the midst of acute rheumatism is not common,[1] while the appearance of this latter in the course of pneumonia, though not unknown, is an event of great rarity.

In the pneumonia of acute rheumatism we sometimes observe that the first stage of the local process is, or seems to be, pretermitted. The lung is discovered to be solid without the preliminary crepitus having been heard. With considerable accord among authors as to the fact there is much difference in accounting for it. It has been maintained that the absence of crepitus is due to the fact that the primary effusion is not in the alveoli but between them in the intervesicular areolar tissue, a statement which, even did it serve, is sufficiently disposed of, as Dr. Waters remarks, by the absence of any such tissue in that situation. It is, perhaps, more to the purpose to observe that the absence of crepitus is by no means confined to this form of the disease. The clinical features of these rheumatic cases are remarkable and suggestive.

The following illustrations of the connection in question may be here interposed.

A girl, nineteen years of age, was admitted into St. George's Hospital for acute articular rheumatism of no great severity, and without heart-disturbance. On the second day she had a fit of alarming dyspnœa, with catching, shallow respiration, pain in the left side and in the affected joints. Repeated auscultation failed to discover any alteration of the heart's action, except in its increased rate. As little could any sounds be detected suggestive of pneumonia, though it must be mentioned that the patient's extreme dyspnœa rendered it impossible to examine the chest very completely. After remaining in great distress from breathlessness for two days, yet free from mental disturbance, this girl died. On post-mortem examination the pulmonary artery, as far as the third and fourth divisions, was found to be filled with decolorised coagulum, and there was a shred of lymph in the right middle cerebral artery. The lower lobe of the left lung is described as 'much solidified from pneumonia, and sinking in water.' The heart was uniformly covered with recent lymph.

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[1] Grisolle speaks of pneumonia as 'one of the rarest phenomena, at least in Paris, of acute articular rheumatism' (*loc. cit.* 425). He notices, however, that English writers do not describe it as very uncommon.

A boy of nine was admitted into the same hospital with acute rheumatism. After eight days' residence he was attacked with 'double pneumonia.' The exact manner and symptoms of this attack need not be given in detail. He recovered from it in a week. He next gets a return of pain in the limbs and alarming dyspnœa; the pulse rises to 120, and there is much visible pulsation of the carotids; free respiration, however, is still heard in the chest. This attack too passes off, and he is up and about again, well, or nearly well. When so far recovered, and with perfectly unembarrassed breathing, he had one night a sudden attack of angina and dyspnœa, which carried him off in a few hours. Here the whole of the right lung, except quite the apex, was red, solid, and airless, 'in the first stage of pneumonia.' The left lung, too, is affected in exactly the same manner, though less uniformly. The pericardium is adherent by recent lymph, the mitral valve very much thickened by fibroid matter.[1]

Both these cases show the main source of danger when pneumonia complicates acute rheumatism, namely, the weakening of the heart due to pericarditis; and no doubt this association is the more grave with a history of previous attacks of rheumatism and consequent heart disease. Yet, even here, provided there is no marked failure of compensation, recovery may be looked for. This is shown in the following case:—

Minnie W., aged twenty-eight, housemaid, was admitted to the Westminster Hospital, October, 1889, with acute rheumatism, having had four previous attacks, the first at sixteen and the last four years ago, when she was in the same hospital, and had at the same time pericarditis, pleurisy and bronchitis.

She has felt the joint pains for ten days, and left work five days ago.

Mother's sister said to have suffered from rheumatic fever, but no other relative.

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[1] See also a case by Andral, 'Clinique Médicale,' iii. 436, and one by Grisolle, p. 426, of double solid pneumonia, associated with acute rheumatism.

An anæmic girl, with acute articular rheumatism of some severity, signs of mitral reflux (organic), and some ventricular dilatation. T. 104°, falling on the second day to 103° ; P. 124, regular ; R. 38 ; white furred tongue.

On the second day dulness and tubular breathing at the base of the left lung indicated pneumonic consolidation. Three days later, *i.e.* on the 27th, small crepitant rhonchus was audible at the right base. T. 103·8, R. 38–45 ; the urine was very acid, giving copious precipitate of urates ; there was no albumen. The rheumatism had yielded somewhat.

On the 29th, that is, five days from the first observation of pneumonia (probably six or seven from its first occurrence) severe diarrhœa set in, by which the patient was much weakened. The temperature fell at the same time, temporarily from over 103° to under 101°, pulse also from 124 to 116. Three days later physical signs of resolution were heard, and by October 3 (nine days from admission), diarrhœa still persisting, the pneumonic signs had almost gone, leaving the articular rheumatism behind.

Analysing the temperature, it was for six days (*i.e.* during the height of the pneumonia) between 103° and 102°, seldom exceeding this range and resembling the sustained temperature of pneumonia and not the erratic tracing of acute rheumatism. After the fall and diarrhœa of the 29th (regarded as a true crisis deformed by the coexisting rheumatic fever) the range was between 102° and 100°, and gradually the tracing assumed the irregular character commonly noticed in acute rheumatism.

Subsequently the girl underwent the usual course of acute rheumatism and made a good recovery, much as though pneumonia had not happened.

The following case is remarkable from the pneumonia, insidious and rapidly developed, appearing to replace the joint affection.

William E., aged 28, carman, healthy, temperate, and well-nourished, but subject to winter cough, was admitted on January 30, 1874. He had been ill for two weeks from getting wet through and remaining in his wet clothes. Stiffness and aching of the limbs had gradually developed into acute rheumatism, affecting at last all the joints. It was the first attack of the kind, and we learnt that in the course of it he had 'fainted' more than once. On admission he had the characteristic aspect of rheumatic fever : profuse acid sweating, flushed and tender wrists and ankles general immobility, a temperature of 102°, highly acid urine, and a tongue already dry and leathery. With the heart's sound no bruit or friction could be heard, but the normal area of superficial dulness was somewhat increased upwards. As for his progress, the temperature remained at and about 103°, the pulse soft and weak 112; perspiration was profuse; he had some pain in his left side on coughing. No further physical signs were observed. Medicinally he was treated with drachm doses of bicarbonate of potash in an effervescent draught, given at frequent intervals until the urine became alkaline (which it did on the third day), and afterwards every fourth hour.

On February 5 the joints had become, somewhat suddenly, almost free from pain, yet the man still sweated profusely, and the tongue was still dry. The alkaline treatment had been continued up to this date. Temperature was now 104°, the highest point yet reached. There was no marked dyspnœa or cough. The heart signs were not notably altered. The patient expressed himself as feeling much better, but except that his joints were set free, neither in his aspect nor general condition was any amendment apparent.

On February 8 (the tenth day) with a morning temperature of 103° (it had been declining daily since the fifth) and total absence of joint pain, though still bedewed with a sour-smelling sweat, this man became actively delirious. In that condition, during a very cold night of midwinter, he got up and partially dressed himself. Delirium of the same restless kind continued at intervals during the following day, and he succeeded more than once in getting out of bed. On February 9, after a night of this sort, he was prostrate and exhausted, with a wandering mind which it was yet easy to recall. Some bronchial catarrh with slight mucous uncoloured spitting was attributable to the exposure; he had no return of joint pain. The temperature had fallen to 102·5. After another night of restless delirium the patient rapidly sank, dying twelve days after admission and probably sixteen days from the acute rheumatic seizure.

The post-mortem examination was made thirty hours after death, when rigor mortis was well marked. In the pericardium there was about an ounce and a half of turbid fluid, with a few shreds of recent lymph. The heart weighed 13 ounces, its left ventricle was firmly contracted, the right ventricle was filled with blood clot. The whole of the valvular apparatus was competent, but a few small vegetations were found on both cusps of the mitral valve. The endocardium generally was opaque and somewhat mottled. The heart substance was soft but not degenerated, and there was slight atheroma at the origin of the aorta, the valves being healthy. The right lung was healthy, and weighed 18¼ ounces.

The lower lobe of the left lung was wholly hepatised, firm and unyielding, red passing into grey so as to give a marbled appearance to a section. It weighed 42 ounces. The surface was covered with thick, shaggy lymph ; the upper lobe compressed (presumably from fluid pressure), the pleural cavity containing much turbid serum. The other organs, all inspected carefully, were healthy.

An exudation rapidly poured into the pulmonary alveoli, and as rapidly consolidating, explains both the suddenness of attack and the exceptional nature of the physical signs which characterise many of these rheumatic cases ;[1] it accounts also for the absence of characteristic sputa, a common observation in this connection, of which we have had repeated experience. Owing to such declensions from the ordinary pattern of pneumonia, no less than to the fact that its proper pyrexia is merged and lost in the pyrexia of the acute rheumatism, attacks of this description are apt to be altogether overlooked.[2]

It is not to be supposed from what has been said that pneumonia in acute rheumatism always happens in this fashion. On the contrary the physical signs which first announce it are often the same as when it occurs alone. But even so the special characters of the disease, in its mode of access and sudden critical remission, are necessarily disfigured by the co-existing rheumatic pyrexia. It is not possible, therefore, that pneumonia in this conjunction should appear altogether in its usual dress, so as always to ensure recognition. Yet, subject to these modifications, it follows the same course, and its resolution is accomplished by the same stages as have already been described for the simple disease.

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[1] In the first volume of the 'St. George's Hospital Reports,' p. 168, in a paper by Dr. John Ogle, there is a woodcut of a preparation which seems to illustrate the pathology of this form of consolidation. It exhibits a section of hepatised lung, with minute branches of the pulmonary artery filled with firm fibrinous coagulum. The specimen is from the Hospital Museum Series vii. No. 10, and is thus described in the Catalogue : 'Specimen showing red hepatisation of the lung, with extensive deposit of dark-red fibrin in the pulmonary artery. Double pleuro-pneumonia had existed, and there was tolerably firm adhesion between the layers of the pericardium ; the cavities of the heart were dilated, especially the left auricle, which was lined by recent yellow fibrin. The margins of the mitral valve cusps were occupied by recent fibrin also, and slight atheroma existed at the root of the aorta.'

[2] The more so inasmuch as pericarditis in such association would certainly be referred to the rheumatism. Should chest pain be complained of at all amid such grievous pain elsewhere, it would direct attention to the heart rather than the lungs, and the discovery of friction would excite no suspicion of pneumonia.

And not only in clinical features does this rheumatic inflammation of lung accord with the definition of acute lobar pneumonia, it may be recognised as well by corresponding anatomical characters. The solid, red or marbled, lung of liver-like consistence, with a surface covered over with recent lymph, as in the cases just detailed, is, as we have already said, no less distinctive.

In this coincidence of rheumatic fever and true pneumonia the occurrence of pericarditis may be referred to either disease. Yet sometimes, without rheumatism, or at least without its common symptoms, an attack of *pericarditis* will be followed by one of pneumonia, as though the two inflammations proceeded from some common origin.

A man of twenty-seven, not intemperate, was admitted into the Burdett Ward of the Westminster Hospital, on April 30, 1874, with pericardial friction and moderate fever, but with neither joint inflammation nor acid sweats. He had long complained of pain in the head and chest, had been much worse during the last two months, and kept from work a week. The rubbing shortly subsided, but not the increased area of cardiac dulness, and on May 18 though imperfectly recovered, the man went out. He returned, however, two days later, having had a rigor in the interval with sharp pain in the left side. His pulse was now 116, respiration 36, and temperature 104·6, the highest point reached.[1] The physical signs now indicated consolidation of the left lung. Total absence of vocal fremitus, with some quavering of the voice, showed at the same time the presence of fluid. The general symptoms were those of pneumonia, yet the aspect was not characteristic, and there was more restlessness and active movement than is usual. On the day before death, which took place on the sixth day of the lung affection, loud pericardial friction became audible, due, as clearly appeared, to renewed contact of roughened surfaces. The man became livid and slightly jaundiced ; the urine albuminous. He died asphyxiated.

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[1] It is observable that although this patient died, and from the first gave no sign whatever of amendment, his respirations rising to 52, the temperature underwent, what would be called from its chart, a critical fall. Reaching its highest point, 104·6, on the second day, it fell continuously

The post-mortem examination, besides exhibiting grey hepatisation of the lower lobe of the left lung, showed on that side a thick layer of pleural lymph. This membrane was closely adherent to the ribs ; externally it was tough and almost leathery, but next the lung puro-fibrinous and soft like butter. The extreme base of the lung had escaped consolidation, and in the pleural cavity was about half-a-pint of straw-coloured serum. The pericardium contained a like quantity of fluid ; its surface was rough with exudation, and displayed the reticular arrangement often seen in pericarditis; some recent shaggy lymph depended from it, but the surfaces were nowhere in union. The right lung was healthy, and no other organ notably diseased.

A limited amount of pericarditis is far from uncommon in pneumonia, and often passes unnoticed. The peculiarity of the present case arises from the fact that the pericardial preceded the pulmonary inflammation, rheumatism being absent.[1]

More often than with rheumatism pneumonia is overlooked owing to the co-existence of *delirium tremens.* Drunkards will rapidly succumb from exhaustion, due, as it seems, solely to that disease, but really the consequence of pneumonia, so insidious as to be discoverable only by physical examination. With no great pyrexia, no cough, no sense of pain or dyspnœa, and no power of spitting, there is little to call attention to the

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till the fifth, when it was below 100°. The day before his death, when the pulse was 148 and respiration 52, the temperature was 100·5. This course of temperature is not rare, and an example of the kind is included in Chap. IX, p. 179, Illustrative Cases.

[1] Pericarditis as a complication of pneumonia is discussed in Chap. VI, p. 106.

chest. The alcoholic poison is supposed to account for the patient's condition, and there seems no more to seek even when a large area of lung is already inflamed. In such circumstances pneumonia runs its course with great rapidity, unperceived and unsuspected. After death, the morbid appearances are very similar to those already described as belonging especially to starvation (Chap. X, p. 194). They differ in no respect from true pneumonia except in the rapid and destructive course of the inflammation.

In view of their clinical history, these cases are described after death as examples of 'latent' pneumonia, and wherever this particular term—not free from objection—is introduced into classification, the classes of patients mainly included under it are the drunken, the destitute, and the subjects of delirium tremens.

From a table of twenty cases of this class examined post-mortem, the following abstracts may be quoted from the Records of St. George's Hospital.

A man, aged thirty-nine, of very dissipated habits, complained some days before admission of pain in the right side, which had been blistered. On admission he was very restless and excited, and was with difficulty kept in bed, his state resembling delirium tremens. After treatment with laudanum he got some hours' sleep, but on awaking, more collected in mind, his face became cyanosed and he very rapidly died.

Post-mortem.—The upper lobe of the right lung was completely solidified in early stage of grey hepatisation. Other organs natural.

A man of thirty-nine, a great drunkard, who had suffered delirium tremens more than once, for two weeks had complained of headache, lassitude, and pain in the limbs, and had taken to bed four days before admission. When admitted he was restless and delirious, but not violent. *No lung affection was recognised*, the man was almost pulseless and died in a few hours.

Post-mortem.—The whole of the two upper lobes of the right lung were in a state of grey hepatisation. The left lung healthy; old adhesions both sides. A few small cysts on surface of kidney.

Of the latency of this form of pneumonia, whether its origin be in alcohol or insufficient food, or a combination of the two, the following is a warning example.

A young woman, whose precise age was not ascertained, was admitted to the Westminster Hospital very prostrate and unable to sit up, without cough or dyspnœa, the pulse very small and weak.

The mother stated that four days before she had been sentenced to four days' imprisonment for drunkenness. She was seen by the prison surgeon, who pronounced her fit for the prison work of oakum picking. This she accordingly did during her incarceration, and according to the matron's statement (which the mother denied) she was able to take the prison diet. On the third morning she again asked to see the doctor, who failed to find anything wrong. On the following day, however, when about to be discharged, she seemed so ill that her mother was sent for to fetch her home. The two came straight from prison to hospital on foot, the girl falling down thrice on the way. Stimulation failed to revive her, and she died about two hours after admission.

Post-mortem.—The lower half of the left lung was in a state of grey hepatisation, and the entire lung sank in water. There was much semi-purulent fluid in left pleural cavity. The right lung was congested and œdematous. The kidneys had finely granular surface but were not otherwise altered, nor were the other organs.

The prognostic significance of alcoholism has been already considered (Chap. VIII, p. 150).

Of the occurrence of pneumonia in patients who are the subjects of chronic disease of the kidneys, and of its frequent fatality and often insidious course in that connection, we have already spoken (Chap. VI); but *acute nephritis* in rare instances concurs with pneumonia. Such an event is hardly sufficiently common to enable us to speak with any confidence from our own experience as to the relative mortality of these cases. Recovery is certainly not rare, and we regard the prognosis as far more favourable than in that more latent form of the disease associated with chronic renal degeneration.

Relapse is not uncommon in these subjects, and the advent of crisis—or rather the temperature fall mistaken for that event

—by no means terminates the period of anxiety. The following abstract may be quoted.

Charles Smith, aged 38, labourer, was admitted to the Westminster Hospital January 1890, a week after rigor and stitch in the left side. There was nothing striking in his personal history save that he had been laid up with 'fever and ague' when abroad. He was said to have had pneumonia five years ago. On admission he exhibited all the characteristic signs of this disease. The middle third of the left lung gave marked tubular breathing, while below this, absent fremitus, distant breathing, and the character of the voice sound indicated fluid. There was some hypertrophy of the heart. Flushed face, furred tongue, and temperature exceeding 103° gave the usual indications of pneumonia.

On the following day (the eighth of disease) the physical signs denoted some increase of lung consolidation, the tubular breathing extending round axilla and now audible below the left nipple. The man's condition, however, was not notably worse, and a marked fall of temperature gave promise of crisis.

On the day succeeding, however (the ninth of illness), there was distinct relapse. The patient was more feeble, and distressed from dyspnœa, the tongue much coated and becoming dry; the physical signs unaltered. The urine (now first carefully examined) contained albumen one third, was slightly smoky and exhibited epithelial and blood casts.

On the eleventh day aspect indicated much distress, he complained of cough and dyspnœa, vomiting had set in, the urine was still albuminous and more markedly smoky, but there was no trace of œdema. The breathing over the left back was less distinctly tubular.

During the next two days the consolidation of the left lung resolved and some pleural friction became audible at the right base. Redux crepitation soon followed, and pleural rubbing was presently to be heard on both sides. The urine ceased to contain blood, but was still highly albuminous and of light specific gravity 1011.

The twelfth day from his rigor, being the fourth of relapse, marked a distinct improvement in the man's general condition. The amount of albumen rapidly decreased, and when he left the hospital after three weeks' residence and a month's illness there was but a faint trace of it, though the urine was still of low specific gravity.

In the extension of consolidation, condition of urine, distinct relapse, vomiting and distaste for food, this case resembles one that followed scarlatinal nephritis, and proved fatal, as related in Chapter X, p. 204,[1] under the heading of Relapsing Pneumonia.

When seen early the symptoms of these patients do not differ from those of ordinary pneumonia. But the very rapid course of the affection, obstinate vomiting, together with active delirium, soon passing into semi coma, will often suggest a renal origin.

A labouring man of twenty-six had been for six days complaining of cough, and had spat a little blood. On admission he had high pyrexia and some delirium, which the same night became active and disturbing to the ward. Physical signs of pneumonia were found at the lower part of the right lung ; the urine, at first suppressed, was found to be albuminous.

By the fourth day the patient had sunk into a typhoid condition, twitching hands and muttering, the tongue black, dry, and furred, and sordes collecting about lips. The sputum from rust-coloured became 'prune juice.' After a temporary rally of two days, obstinate and constant vomiting came on, and he died on the eleventh day from admission.

Post-mortem.—The kidneys were large and solid with yellow increased cortex and mottled surface.

The whole of the right lung was completely hepatised, grey ; breaking down in several parts into small 'abscesses' ; the right pleura was adherent. The pericardium coated with a thick layer of recent lymph. The left lung was natural.

Pneumonia, pursuing a like fatal course, will sometimes appear quite suddenly in the course of acute nephritis, as in the following case.

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[1] Acute nephritis, even when apparently recovered from, must largely qualify the otherwise favourable prognosis of lobar pneumonia in young children. The following example may be quoted to that effect. A child of two years was admitted with history of scarlatina a month before, followed, as was thought, by recovery. Two weeks later the parents noticed convulsive twitching. Dyspnœa appeared only a day before admission, with stridulous breathing. The child was already collapsed, and died in a few hours. Post-mortem, the kidneys were pale, smooth and mottled, the cortex increased. The greater part of the right lung was solid, the reddish-brown mottled with grey hepatisation ; there was lymph covering the pleura. The left lung had some spots of consolidation, but the left pleura was natural. There were some specks of recent lymph on the mitral and aortic valves.

A man of fifty had had good health until two months before admission, when he got œdema of face and legs with cough, the urine becoming scanty and bloody. After four days in hospital the dropsy subsided, but urine continued albuminous. On the seventh day the sputum became blood-tinged and pulse frequent; the patient shortly after became very livid and he died after eleven days' residence.

Post-mortem.—Kidneys were large, smooth, with ecchymosis throughout the cortex, weight 16½ ounces; heart was hypertrophied and its left ventricle much thickened, weight 23 ounces; there was some atheroma of the aortic valves. The whole of the left lung, except apex, was consolidated, some thick recent lymph covered the left pleura, whose cavity held much turbid fluid.

(The two preceding cases are from Notes in the Medical Register, St. George's Hospital.)

Of *meningitis* and other acute affections we have spoken (Chapter VI, p. 119). They are to be regarded rather in the light of complications than associates of pneumonia. Some cerebral symptoms apt to be mistaken for meningitis in the case of children, will be described in the next chapter.

Pneumonia is commonly spoken of as concurring with *continued fever, both enteric and typhus*, and no doubt pulmonary consolidation may occur in both, sometimes hypostatic sometimes lobular, and contribute directly to the death of the patient. Whether or not the lung condition is fitly described as intercurrent pneumonia may admit of question. The expression, though freely used in clinical records of these fevers, is seldom borne out by the post-mortem description. 'True pneumonia,' wrote Dr. Murchison,[1] 'with exudation of solid lymph, is comparatively rare in typhus. The majority of cases of so-called pneumonia are examples of hypostatic consolidation with bronchial catarrh. It is not always easy to distinguish it during life from pulmonary hypostasis, and in fact the lesions may exist together. As for pleurisy, its advent is latent, and the effusion almost always fluid.' It is added that when true pneumonia does occur it is sometimes 'developed with great rapidity, the whole of one lung becomes solidified in a few hours. Such cases may terminate in gangrene.' 'I have never chanced,' says Dr. Murchison, 'to meet true pneumonia of the apex as a complication of typhus. As a rule the consolidation has been in some part of the lower lobe, and often it has been lobular.'

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[1] 'On Continued Fevers,' p. 184.

Of enteric fever, the same author writes that pneumonia is more common than in typhus, occurring in 13 out of 100 of his cases, but the important qualification follows, that in this small percentage the form is usually lobular. Two illustrations are given of the occurrence of this so-called pneumonia in enteric fever. In the first, a boy of eleven, the lungs showed isolated nodules scattered throughout both, friable and scarcely crepitant, but not granular on section. The right lung weighed 12, the left 13 ounces. The pleuræ are not mentioned. In the second patient, a youth of nineteen, both lungs contain circumscribed nodules of granular consolidation, varying in size from a pea to a walnut, the right lung weighed 30, the left 24 ounces. Again the pleuræ are not mentioned.

Turning to our own records, the post-mortem evidence of twenty years (1848–1867, which includes the typhus epidemic of 1866), at St. George's Hospital, furnishes but one example of lobar pneumonia in connection with typhus, and even this case, which was under the observation of one of us, then medical registrar, is ambiguous, inasmuch as there was no accompanying pleurisy.

A woman of thirty-six, well-nourished and in good condition, was admitted July 28, 1862, stating that a week before she had been seized with shivering and acute aching catching pain in right side;

cough and dyspnœa at the same time. On admission the aspect was distressed and anxious, face flushed, respirations 52, pulse 120, very feeble; skin was hot and dry, tongue dry and brown. Breathing over right chest was harsh and indistinct, but not tubular nor even markedly bronchial. A few moist sounds were audible at the left apex, but respiration over that lung was otherwise healthy, while both sides of chest were equally resonant. There was no expectoration.

On the second day delirium set in, and an indistinct mulberry rash was visible ; breathing over the right side was still imperfect and more bronchial, but not tubular. Delirium continued through the following day, sordes collected about teeth, the eruption did not further develop. In the view that the patient was suffering from typhus, prevalent at the time, she was treated with large doses of quinine, more than 80 grains being swallowed in twenty-four hours, by which time the skin was cool, and pulse 72, while the respirations were 60. At the instant these observations were taken the colour faded from the woman's face and she immediately died, having been in hospital four days and ill for eleven days.

Post-mortem.—With the exception of very slight atheroma affecting aorta and mitral valve, the organs were normal. Large and small intestines unaffected. Liver and kidneys healthy; spleen soft. Both cardiac ventricles were uncontracted, the left containing fluid blood with some specks of black coagulum. The right ventricle, also uncontracted, contained a decolourized clot of great length and tenacity extending into the auricle and cavæ.

The whole of the right lung was in a state of grey hepatisation, except a small portion near the apex, and a small patch at the lower margin. Texture was grey and mottled, and in parts softened as if on the point of breaking down. Pus could be readily squeezed out. The bronchi were congested and contained frothy fluid. The pleura was natural. The left lung was healthy but for two minute cretaceous tubercles.

Hepatisation after this form is certainly very rare in typhus, and we do not find a case to match it. Not less exceptional is the occurrence, under whatever circumstances, of a lobar consolidation having all the characters of pneumonia yet with no accompanying pleurisy. It is to be remembered, also, that the diagnosis of typhus was not fully established. In whatever light pneumonia is to be regarded, whether as a complication of typhus, or a primary hepatisation without pleurisy, it is alike anomalous.

In like manner lobar pneumonia is, in our experience, extremely rare in enteric fever, although, as in the case of typhus, secondary lobular consolidation affecting both lungs is not uncommon. This latter form of inflammation occurs very insidiously in the third week or later, the blunted faculties of the patient, absence of expectoration, and difficulties in the way of physical examination concurring to conceal it. With children, indeed, where, as we shall see, lobular pneumonia is occasionally nearly associated with lobar (Chapter XII), enteric fever may exhibit in rare instances[1] the two forms in combination.

William M., aged 10, died in the Westminster Hospital of enteric fever in the third week and without open signs of pneumonia.

Post-mortem.—There was the usual ulceration of Peyer's patches indicative of the age of the fever. But the immediate cause of death was undoubtedly hepatisation of the lower lobe of the left lung in the grey stage, and with recent pleuritis. Yet, neighbouring upon this true pneumonia, and affecting both lungs, were irregular patches of carnified lung, numerous nodules containing dilated bronchi filled with pus, general bronchitis, and some œdema, these latter representing not infrequent complications of the disease in question.

In rare instances pneumonia will occur so early in the course of enteric fever as to mask the symptoms of the latter disease. In the following case we have acute pneumonia (right lung) with the onset of enteric fever.

James M., aged 25, a footman, admitted into the Middlesex Hospital on October 22, 1881, on the sixth day of an illness which commenced abruptly with vomiting and rigors, followed by diarrhœa, pain in the right side and dyspnœa. He attributed the attack to a chill gained whilst out shooting with his master in Yorkshire during the previous three weeks.

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[1] Henoch, quoting the case of a girl of twelve with typhoid fever dying of lobar pneumonia, speaks of it as 'the most astonishing case I have had.' 'Lectures on Children's Diseases,' vol. i. p. 402.

On admission his temperature was 104°, pulse 108, respirations 36. He was a well-nourished, muscular man, the face slightly dusky, flushed, lips dry, *alæ nasi* working. The tongue was coated; the breathing was shallow, chiefly diaphragmatic. There was dulness on percussion over the right front, from the third rib downwards, and posteriorly from the scapula to the base of the lung. The resonance at the apex was high-pitched; the left side was normally resonant. The breath sounds over the dull area in front were markedly tubular; behind they were weak but free from râle. On the left side the breathing was normal; the first sound of the heart was weak, the second prolonged. The spleen was not apparently enlarged, but the hepatic area reached about two inches below the costal margin.

A quinine mixture was prescribed, and cold compresses were applied to the right lung.

On the 23rd the tubular character of the breath sound over the right front was more plainly marked, especially in mammary region. In the axilla from the sixth rib there was weak breathing with some crepitation. No change in the signs posteriorly. The skin was dry and hot. The temperature, which had fallen to 100·8 at 7 A.M., rose to 103·8 at 9 A.M. and to 104° at 5 P.M.; the pulse was dicrotic; the face was more dusky, the lips livid; respiration 36.

October 24.—Tubular breathing less marked, but still some fine crepitation over the dull area; considerable dyspnœa. Expectoration scanty and rusty. The bowels were opened three times. The pyrexia was still high, temperature ranging between 104° and 102·8.

October 25.—Maximum temperature 103·6, pulse small and compressible, 104, respiration 32. The resonance over the right front is improving, and the breathing is no longer tubular, but behind it is more bronchial in quality than hitherto. There is more and coarser crepitation in the axilla, with some musical rhonchus. The urine is free from albumen.

October 26.—Tongue thickly coated. Temperature 103·4, pulse 112, respiration 30.

The area of dulness now gradually diminished, together with the other signs of consolidation, but the pyrexia did not abate. Indeed, on the 27th the temperature rose to 104·2, and on this and the following day the patient was given a bath which slightly controlled the fever. On the 29th, although the face was still dusky, the sputa were simply mucoid; there was crepitation over the whole of the right lower lobe. And now over the abdomen there had appeared some rose-coloured spots, the first crop of which had been noted on the 27th, the eleventh day of his illness. There was also slight diarrhœa, and the motions were becoming more characteristically typhoidal. These facts, together with the continuance of the pyrexia, for which frequent sponging and the administration of large doses of quinine was prescribed, left no doubt that the case was really one of enteric fever, the onset of which was accompanied by pneumonia. Rose spots continued to appear in crops until the seventeenth day, when there was a profuse eruption of sudamina. The pulmonary signs gradually cleared up, and the subsequent course of the fever was uncomplicated ; the most marked feature was profuse sweating. The evening temperature did not become normal until November 11, and convalescence was then uninterrupted. It should be added that at the end of November the breath sounds at the base of the right lung were still weak, but there were no râles, and there was good resonance all over the lung.

He left the hospital on December 6.

This case illustrates a very rare concomitance. Murchison observes ('Continued Fever,' 3rd edit. p. 557) : 'Pneumonia rarely supervenes before the third or fourth week [of enteric fever] ; in rare cases it occurs early in the attack, and may be mistaken for the primary disease.'

A similar account has to be given of the infectious fevers of early life, *scarlatina*, *measles*, *diphtheria*. In measles, as is well known, broncho-pneumonia is very common, but the inflammation is almost always consecutive to bronchitis, two-sided, and without fibrinous pleurisy. Lobar pneumonia is uncommon. In scarlatina, according to Dr. West, 'pneumonia is a more frequent affection than pericarditis, running its course without any marked symptoms, though a large portion of one or both lungs may be found after death in a state of hepatisation.'[1]

Other authors make little mention of lobar pneumonia in this connection. It is not to be forgotten that of late years the opportunities for studying scarlatina have been much curtailed by the operation of the now universal law excluding it from general hospitals. That lobar pneumonia is not common in scarlatina may be taken as certain ; but considering the connection of that fever with pericarditis, nephritis, and rheumatism, it would be of interest to have more accurate knowledge as to its frequency.

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[1] 'Diseases of Childhood, p. 727, 4th edition.

We are better informed, however, in the case of *diphtheria.* A large number of deaths in this disease, especially in the younger children, are chargeable to secondary inflammation within the lungs, to bronchitis, broncho-pneumonia, and lobar pneumonia in that order of frequency.

SUMMARY OF 100 FATAL CASES OF DIPHTHERIA, IN WHICH THE LUNG CONDITION WAS ASCERTAINED BY POST-MORTEM EXAMINATION, FROM JULY 29, 1884, TO NOVEMBER 1889, HOSPITAL FOR SICK CHILDREN.

Lungs natural, or with no marked lesion	Lungs congested	Lobular pneumonia	Lobar pneumonia	Other states: Collapse—emphysema—Empyema &c.
20	11	20	14	35

CONDITION OF LUNGS IN 60 OF THE ABOVE IN WHOM TRACHEOTOMY WAS PERFORMED.

Lungs natural, or with no marked lesion	Lungs congested	Lobular pneumonia	Lobar pneumonia	Other states: Collapse—emphysema—Empyema &c.
13	4	10	13	20

In the great majority of the latter death followed the operation so quickly that the condition of the lungs could hardly have been influenced by this procedure.

There is difficulty also in separating lobar from lobular pneumonia, and the former seldom occurs alone. But taking three of the best-marked cases of lobar pneumonia, we get the following : one, a child of twelve months (27), died nineteen days after tracheotomy ; a second, aged four (86), twenty-four days after it ; a third (less distinctive than the other two), also aged four (88), eleven days after. In all of these the pneumonia is in an early stage, indicating a considerable interval between its commencement and the operation of tracheotomy. There are but two other examples (6 and 43) of children surviving the operation for more than a week, and then dying of lung complications, and both died of lobular pneumonia ; one (6), aged three, thirty-three days after tracheotomy, the lung affection being 'recent' ; and the other (43), aged one year, twenty-two days after. In this last the broncho-pneumonia affected one lung only, thus partaking the character of lobar. Besides these, there is only one patient of the 100 exhibiting typical lobar pneumonia, apart from tracheotomy (59), an infant of twenty months.

Speaking generally, it appears that collapse, with emphysema and broncho-pneumonia, are the chief morbid appearances in death from diphtheria, whether tracheotomy has been performed or not ; while lobar pneumonia (although not confined to such cases) shows chiefly in patients who have undergone that operation, and apparently recovered. The conclusion seems to be, not only that liability to lung inflammation is to be reckoned among the after-perils of diphtheria, which is notorious, but that such liability continues long after tracheotomy and the termination of the disease, so far as the throat is concerned ; while true lobar pneumonia, so little to be dreaded in young children, in other circumstances, is very often fatal in this connection.

CHAPTER XII

PNEUMONIA IN CHILDREN

Association with broncho-pneumonia—Prominence of nervous symptoms—Delay and obscurity of physical signs—Diagnosis—Etiology—Rate of mortality—Essential points of distinction from broncho-pneumonia—Chronic pneumonia—Empyema—Some points of treatment.

In early childhood, as has been said, pneumonia presents the essential characters of the disease as it is seen in adults—sudden onset, limited duration, and quick recovery. But in infants and young children it is far more common to meet with lung inflammation secondary to bronchitis—an affection of indefinite duration, variable course, and high mortality. Widely, however, as does broncho-pneumonia differ from lobar, yet between these two forms of lung inflammation there are numerous gradations, so that it is sometimes difficult or impossible in particular instances to distinguish, whether from physical or other signs, the one from the other. Moreover, even in the most strongly-contrasted examples of lobar and broncho-pneumonia respectively, there are certain common histological factors, which we have already described under morbid anatomy.

The following example well illustrates some of the peculiarities of child's pneumonia.

Thomas F., a light-haired, strumous-looking child, aged 6, was admitted into the Westminster Hospital with a history of sudden illness, commencing the day before, on his return from school. He then complained of epigastric pain, and shortly after became unconscious. On admission the child was in a state of extreme restlessness, with widely-dilated pupils, terrified aspect, and exhibiting all the symptoms of active delirium within a child's reach. Temperature 104·8, pulse 164, respirations 41. It was with extreme difficulty that he could be got to swallow. He continued delirious and sleepless, with temperature varying between 101° and 104°, for three days. *On the fourth day, for the first time*, small inspiratory crepitus with tubular breathing became audible at the base of the right lung. From that time, though the temperature rose and fell during the next two days, the nervous symptoms abated, and the case assumed the ordinary aspect of pneumonia. On the ninth day redux crepitation was heard, and the child made a rapid recovery, as might have been anticipated so soon as the nature of its illness was revealed.

More common than delirium, however, at the onset of child's pneumonia, is convulsion, and, commonest of all, vomiting; while in many cases diarrhœa is present in place of constipation. Such symptoms, as is well known, are the same as usher in most of the acute affections of childhood; and where the physical signs of pneumonia are delayed, as in the case just quoted, it is only by the observation of the temperature, pulse, and respiration, that we can discriminate the disease from many others. Yet it is only at the outset that this obscurity obtains. In its suddenness, high temperature, critical end, short convalescence, and small mortality, the pneumonia of childhood corresponds closely with that of later life.

The initial symptoms of lobar pneumonia in childhood will be best displayed by an actual analysis of cases. Taking twenty well-marked examples of the disease occurring at the Hospital for Sick Children, within the last eighteen months, under the care of one of us, we find the average age between four and five years. Twelve are between one and three, 3 are between three and nine, and 5 are children of eleven and twelve. The most constant of all amongst the early symptoms was vomiting, which was severe and repeated in 14, only slight in 1, and occurring late, that is during crisis, in 1; thus leaving but 4 exempt. Convulsion, on the contrary, was only noted as a commencing symptom in 5—all young children. In one of these, an infant of six months, there was also strabismus, and the condition closely resembled meningitis. It had crisis on the seventh day. Only two of the twenty had rigors—a child of eleven and one of four-and-a-half. Premonitory symptoms preceded the actual onset of pneumonia by several days in as many as 7 patients; such were diarrhœa, bronchial catarrh, convulsion, drowsiness. This last symptom, indeed, in some degree, was seldom absent. Drowsiness is a very characteristic symptom in the pneumonia of children, and is, no doubt, analogous to the listless 'typhoid' state of adult pneumonia. Only 1 case was fatal, and that, not from pneumonia, but from the accident of contracting diphtheria. The rest all underwent favourable crisis, and none were gravely ill for over a week. Thus, as has been said, in temperature, in duration, and in spontaneous disposition to recover, these patients resemble their elders. A stormy onset, with high fever, calming down in two or three days upon the development of the lung inflammation, and subsiding altogether, with critical temperature fall, in about a week—that is the short description of child's pneumonia. In some instances, indeed, the pyrexia does not last over two or three days, while the local signs, often of very limited area, do not become distinct until after the crisis.

There are other peculiarities of childhood, besides nervous instability, that may alter the pattern of pneumonia as seen in adult patients. Amongst these is the common sympathy of the mucous membranes in early life. Just as diarrhœa is a frequent accompaniment of child's bronchitis, so in pneumonia both the pulmonary and intestinal mucous membranes may sympathise, giving rise both to diarrhœa and general bronchitis. If to such bronchitis a broncho-pneumonia succeed—an event, as we shall see, the more common the younger the child—we reach then a mixed condition, where, unless the patient has been observed from the first, precise diagnosis is impossible.

In the following case, a delicate child, subject to bronchial catarrh, contracted pneumonia. On its defervescence a broncho-pneumonia succeeded; and this, while it delayed recovery, gave rise to a combination of physical signs which, but for the early observation of the case, would have been difficult to interpret.

Hannah A., aged 9, a delicate child, who had lately suffered from bronchial catarrh, but was at the time in fair health, was admitted into the Westminster Hospital June 8, 1889, on account of a fit two days before, lasting twenty minutes, and with general convulsion. There was no vomiting, but some diarrhœa followed the fit and cough increased. On admission the aspect is not unnatural, and the child voluntarily sits up in bed. But respiration is hurried, 44, *alæ nasi* in movement; temperature 104°, pulse 120, the tongue white furred. Physical examination reveals anteriorly a patch of solid lung above right nipple.

The mother states that the child is subject to winter cough, apt to perspire at night, and that two years ago she had three fits in succession.

The day following admission, after a restless and delirious night, the patient was flushed, and in every way worse. Temperature 104°, pulse 140, respirations still 44. Slight jaundice, with diarrhœa and light-coloured motions. She also vomited once or twice, but takes nourishment well. The signs of consolidation are still well marked, but some crepitant rhonchus is now heard, which, with the lessened intensity of tubular breathing, seems to indicate commencement of resolution. The morbid sounds are confined to this lung.

For the next three days the child was extremely prostrate, with short, painful cough, moaning respiration (56–68), the pulse 130, temperature 103°, much night delirium, the face becoming dusky. On June 12 (six days from the fit) profuse sweating occurred, the temperature fell critically to normal, but without general improvement; on the contrary, the respirations at 8 P.M. of that day reached 80. It was now observed that the area of crepitant rhonchus had enlarged, that tubular breathing was no longer audible, and that crepitation could be now heard at the *left* apex. The cough had become loose, with abundant mucous expectoration. On the 14th (or ninth day) occurred the real crisis, with much diarrhœa ; temperature, which had again risen, falling once more to normal, while the flushing of cheeks subsided, and the aspect became natural. There was still, however, considerable dyspnœa, respirations 72, dropping by midnight to 52, loose cough, and much mucous expectoration. And now over both lungs crepitant rhonchi were audible, those of the right side being of the higher pitch, and some percussion dulness remaining. On the 19th (thirteenth of illness) the patient was without fever, with good appetite and clean tongue, respirations 36, and pulse 108. Cough still continued, but the expectoration was less, while the signs of general bronchitis persisted. A week later the child was convalescent, though some signs of general bronchitis still remained.

Again, physical signs are not wholly trustworthy in determining whether lung consolidation be lobar or lobular. Solidified patches lying close together throughout a single lobe are not distinguishable by the ear from a uniform area of lobar consolidation. It is true that the absence of general bronchitis, and the fact that the morbid sounds affect one lung alone, will tell so far in favour of lobar, rather than broncho-pneumonia; true, also, that a sustained high temperature suggests the same inference ; but these signs are inconclusive, and in infants more particularly (with whom fatal lobar pneumonia is certainly rare), the mere fact of death will of itself raise the probability that the affection, whatever its clinical symptoms, is in fact broncho-pneumonia.

How nearly lobar may resemble lobular pneumonia in its mode of onset, temperature and physical signs, appears from the following :

Frances M., ten months, was admitted into the Westminster Hospital, June 4, 1889. Two days ago the child became feverish and drowsy, diarrhœa set in the same day, the motions being green and slimy. There was no sickness. A day later (*i.e.* the one before admission) the child was convulsed two or three times ; it had also some delirium during the night. On the morning of the 4th, the day of admission, the patient became much worse ; drowsy, hot, very thirsty, and constantly screaming. The mother also noticed that the child was very short of breath, and that the nostrils dilated.

The previous history is to the effect that the child was well and healthy up to three months ago, when it had bronchitis. It was weaned at two months, and bottle-fed until a month ago. Since that fed on bread and milk, but no meat. An only child, born at full time, father healthy, mother said to suffer from 'bronchitis.'

Admission temperature is 105°, respiration 70, pulse 168. The lower lobe of the right lung yields dulness and tubular breathing; the left lung is free.

No more need be related for the present purpose than that the child grew worse, diarrhœa continued, and the respirations on the 6th (fifth day of illness) reached 80. On the 8th (seventh day of illness) the child died; the day previously some crepitant rhonchus having been audible at the base of the *left* and hitherto unaffected lung. The temperature tracing was not of the intermitting kind characteristic of broncho-pneumonia, but of the sustained kind as in lobar pneumonia. That is to say, its range was between 105° and ·103·5, and but once at mid-day of the one preceding death fell as low as 102°.

We have thus singularly blended the symptoms of lobar and of lobular pneumonia. The physical signs, limitation to one lung, and course of temperature of the one; the antecedent bronchitis, tender age and, later on, implication of the fellow lung of the other. Had the child recovered the diagnosis would have remained in doubt, but the mere fact of death went far to strengthen the belief that the inflammation was lobular. This, on post-mortem examination, proved to be the case.

The lower three-fourths of the right lung and the lower half of the left were indurated and purple in colour. On section the consolidated portions showed dilatation of the bronchi and numerous white islets. There was no caseation or breaking down; much fluid in pleural sacs. Vicarious emphysema at upper parts of both lungs. (P.M. Vol. V., No. 183.)

Not only may temperature be sustained in broncho-pneumonia so as to resemble precisely the tracing of the lobar form, but true pneumonia may be still further imitated in rare instances by the occurrence of crisis.[1] Thus, a child of sixteen months was admitted to the Westminster Hospital with a history of bronchitis and diarrhœa, symptoms of some duration, but which had increased in severity during the two preceding days. The physical signs were those of general bronchitis and broncho-pneumonia, and on the fourth day from admission temperature rose to 105°, pulse was 180, respirations between 60 and 70. For ten days more the temperature range was between 103° and 105°, diarrhœa continued, and the child was extremely prostrate. On the fourteenth day from admission diarrhœa ceased, there was profuse sweating, and temperature fell from 104·5 to 97°. In a week from this critical amendment the child was convalescent.

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[1] The occurrence of crisis in broncho-pneumonia is denied by Henoch, but admitted by implication by most other writers.

The importance of diagnosis in such cases as these is no less obvious than its difficulty. For if we can determine that a child's pneumonia is lobar, we know almost for certain that it will shortly recover, provided it be primary, while nothing is more uncertain than the duration and final issue of broncho-pneumonia. Admitting that the example just quoted is one of unusual difficulty, the practical rule would seem to be this: A sudden onset, a sustained temperature exceeding 103°, and a single seat of consolidation in one or the other lung, are symptoms in favour of a lobar or recovering pneumonia, and in children over seven or eight years, may with confidence be thus interpreted. But in babies and young children we have far less assurance. Much will depend upon the antecedent history, and should it appear that the pneumonia has developed out of a preceding bronchitis no confident prognosis is possible.

In speaking presently of broncho-pneumonia we shall have occasion to mention other points of resemblance between these two forms of lung inflammation, while at the same time pointing out the wide and fundamental distinction between them. We have now to complete the clinical account of lobar pneumonia in childhood, in so far as it differs from the same affection in adults.

These points of difference have reference mainly to certain peculiarities in the physical signs; to the near likeness of pneumonia to some of the specific fevers of early life; to the instability of the child's temperature and liability to recurrence of its pyrexia, and sometimes of the lung inflammation itself. Yet in regard to all these phenomena it would be more true to say that the child exhibits them more than the adult, than that the child alone exhibits them. Certain features of the disease which are commonly seen in early life disappear or become rare later on, but the main characters of pneumonia are apparent at all ages.

The latency or delay of the proper physical signs which, together with the ambiguous nervous phenomena, contributes to the obscurity of diagnosis in many cases, has been attributed to the gradual extension of the inflammation from centre to periphery, that is, from inaudible to audible parts of the lung. We have already discussed this point in connection with the physical phenomena of the disease (Chap. X, p. 192). Be the explanation what it may, it is certain that as many as six days may elapse before these signs are fully developed, and that without careful and frequent observation they may be missed altogether. The earliest physical sign of all is weakness of vesicular breathing at the part affected, the next, fine (more or less) inspiratory crepitus heard only at the end of inspiration, while the neighbouring region is apt to yield to percussion a higher note than elsewhere. In infants, as is well remarked by Dr. Fagge, 'a bronchophonic cry is often the only auscultatory sign that can be obtained.' Indications such as these, especially when confined to apex or axilla, are likely to be overlooked, and thus the real interval between the invasion and its earliest local expression may seem longer than it is. Meanwhile drowsiness, vomiting, perhaps convulsion and even strabismus, symptoms unexplained by teething, improper food or any other eccentric irritation (matters which, it need hardly be said, must be rigidly investigated), do not always suggest what is impending. In adults, on the contrary, we recognise pneumonia almost for certain before the local signs appear. Yet, however obscure the commencement of the child's disease—an obscurity which, as we shall see, is not without some glimmering of light—nothing can be plainer than its course and progress when fully developed;[1] and we cannot subscribe to the statement of Professor Henoch[2] that 'cases of supposed recovery from meningitis are really cerebral pneumonia wrongly diagnosed.'

A further difficulty in diagnosis may arise from the early *resemblance of pneumonia to scarlatina.* This appears not only in the pungent dry skin and high fever, but sometimes in notable congestion of the pharynx and slight redness of skin. This latter symptom never, so far as we know, has the punctiform vivid colouration of well-marked scarlatinal rash, but rather resembles, as Henoch observes, the patchy reddening of surface seen in meningitis. Yet to scarlatina with ill-developed eruption, and to meningitis, the lobar pneumonia of early childhood may offer so near a likeness that no immediate diagnosis is possible.

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[1] This statement, however, is not without exception, as the following case shows:

Beatrice C., an infant of five months, was under the care of one of us at the Hospital for Sick Children, October 21, 1889, with a history of diarrhœa and wasting of two months' duration, and had obviously been insufficiently and badly fed. The child was pale and wasted, with depressed fontanelle and head retraction. There was some fine crackling inspiration generally over chest, but nothing more distinctive, there was no cough and little pyrexia. During the five days intervening between admission and death attention was wholly occupied with the well-marked nervous symptoms, retraction of head to an extreme degree, twitching of face muscles, rigidity with forcible flexure of thumbs on palms. Only on the day of death and when cyanosed, was dulness detected at both bases with bronchial breathing and some fine crepitations. The case was regarded as one of basic meningitis. Post-mortem, however, nothing morbid was found in the brain, but both lungs were solid as to their lower lobes. Upper parts of both were slightly emphysematous, and the lower lobes in state of red hepatisation; there was no pleurisy and no tubercle; and the mesenteric glands with the rest of the organs were normal, except that the liver was slightly 'nutmeg.' 'Post-mortem Book,' Hospital for Sick Children, vol. viii. p. 237.

[2] 'Lectures on Children's Diseases,' vol. i. p. 405. Sydenham Society.

In this difficulty the cough, the dyspnœa, and the earliest physical indication of lung change are the main points to which attention should be directed. Yet it must be remembered that dyspnœa of some degree is never absent from pyrexia; that irregularity of respiration is met with in meningitis as well as in pneumonia; and that slightly bronchial respiration over apex and upper sternum in babies and young children is of itself an ambiguous sign. It is *the degree* of dyspnœa, and the character of the chest movements therewith, along with the disturbed pulse respiration ratio, that often give the first intimation of the truth. Especially significant is the slight indrawing of the lower intercostal spaces and epigastrium, the short cough that from time to time interrupts inspiration, and the movement of the alæ nasi. With this the fact that the lung is *silent* over one spot, or that the breathing is bronchial in a part away from the large bronchi, or that there is an inspiration crepitus over ever so small an area, and of very limited duration, deposes almost with certainty in favour of pneumonia. The slight, changeful and transitory character of its physical signs, and the want of harmony between the severity of attack and the area of lung involved, are striking features of the disease in early life. It may be added that the site of the inflammation as between upper and lower lobe exercises but little influence, and some apex pneumonias in children have their crisis on the third or fourth day.[1]

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[1] Dr. Eustace Smith is of opinion that apex pneumonia is especially short and favourable. 'Diseases in Children,' p. 425. 'The Collective

The following is among many examples of ill-expressed physical signs concurring with severe and typical pneumonia :—

Sophia G., aged one year and eleven months, admitted into the Westminster Hospital, June 6, 1889. She was in perfect health till June 4, two days before. On that night, when in bed, she sweated profusely shivered and vomited several times, felt very hot but as yet had no cough. A cough, the remains of pertussis contracted in March, had left her just before this acute attack. On admission pulse was 148, respiration 60 (quickening to 72 on the sixth day), temperature 105°. The child had some diarrhœa, and was occasionally sick. The breathing at first was somewhat harsh and bronchial at the left base, and a few moist sounds were audible in front. *These signs, which were never distinct, did not persist.* The temperature however was maintained, and in spite of frequent sponging recorded 105° each day until the seventh of illness. *On the 13th (ninth day of illness)*, when temperature had fallen to 104°, but vomiting and diarrhœa were still urgent, *bronchial breathing was audible at the right apex*, with impaired resonance and marked increase of voice conduction. From this day the temperature gradually fell, and on the 14th (tenth day) temperature was 100°, pulse 120, respiration 28. The physical signs, however, developed no further, and on the 20th (seventeenth day) the child was convalescent.[1]

Some other characters of pneumonia in children, such as its occurrence sometimes in erysipelatous form and extension from its first seat so as gradually to involve a large area of one or of both lungs—the 'pneumonia migrans' of some authors—do not need separate notice here, since the very same thing is met with in adults. It needs mention, however, that whenever a child's lobar pneumonia seems thus to spread, and especially when it comes to implicate the fellow lung, it is necessary to discriminate between this spreading form of true pneumonia and the supervention of broncho-pneumonia—an

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Investigation Report' (page 46) would lead to the conclusion, corresponding with our own experience, that this locality of the lung is more commonly invaded in children than in adults.

[1] In this case, and in another mentioned in the analysis on page 231, vomiting and diarrhœa are among the symptoms of crisis.

event, as has been said, not uncommon with the younger patients through the intervention of bronchitis.

Certain *peculiarities of temperature*, such as the ill-sustained fever with delusive descents apt to be mistaken for crisis, and ephemeral pyrexia during the period of convalescence, are not to be reckoned as peculiarities of child's pneumonia, but rather as characteristic of pyrexia in early life.

It has been said that the signs of lobar pneumonia in young children may be so intermixed with those of nervous disturbance, that it is impossible from the history to fix the precise date of origin of the former. Sometimes, no doubt, the disease is recurrent ; but what is very striking, and but little noticed in books, is the *occurrence of pneumonia as the final event which brings to a term a long series of nervous symptoms* which, at the time, have been variously interpreted.

Amelia M., aged sixteen months, was admitted to the Hospital for Sick Children, November 14, 1888. On October 27 (eighteen days before), when in good health, she was taken out in the rain and noticed to shiver, was very feverish thereupon, and had much dyspnœa. On October 31 (fifteen days ago) she suddenly became very drowsy, and had vomiting and diarrhœa. *General convulsions then set in and continued for ten days*, slight at first but gradually increasing, and at last severe. The child then began to mend, but up to time of admission still had slight convulsion. On November 14 she was thought sufficiently improved for removal, and was accordingly brought to the hospital as an in-patient

The patient is of healthy family, and five other children are living. Before the supposed chill the child suffered from painful dentition. She is well nourished, but extremely pale ; T. 103·5, R. 64, P. 160 ; restless and very prostrate, skin extremely dry and pungent, some sordes are on lips, and the tongue wants moisture. There are physical signs of consolidation of the upper part of the left lung.

After three days' residence, the signs, physical and other, undergoing little change, this child's temperature fell critically from 103° to 96·5 in twelve hours, and it did not again ascend above the normal. Yet there was no marked improvement until four days later (November 20), when the consolidation began to resolve and the child rapidly got well.

Thus the sudden fall of temperature was 21 days from the chill and 18 days from the first severe nervous symptoms, but the real crisis with recovery was twenty-five days from one and twenty-two days from the other.[1]

It is not to be supposed, from what has been said, that the onset of pneumonia in young children is always obscured by nervous phenomena preceding its local signs.[2] The lung inflammation is often obvious from the first, and, except for the special manifestations of the fibrile state proper to childhood, the disease is of the form we see in the adult. And with the older children, those let us say who are over eight, the not infrequent difficulty of diagnosis between the two forms of pneumonia met with in the younger subjects in large measure disappears. Lobular pneumonia becomes far less common, and lobar assumes most of the characters that it wears in later life. Yet for some years later the occurrence of vomiting and diarrhœa at the onset, rather than of rigor with constipation, a notable drowsiness and sometimes convulsion, in place of the blunted perception and night muttering of the adult, are still features in child's pneumonia. More often too is a marked fall of temperature found to precede and not to accompany critical amendment, while after this event (which is often signalised by profuse sweating), ephemeral pyrexia, of no special significance or harm, may occur from time to time for days or even weeks.

The *etiology* of pneumonia in children is involved in much obscurity, and needs further and independent study. 'The

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[1] See Dr. Eustace Smith (p. 426, *loc. cit.*), who relates the case of a child of three, showing nervous symptoms for five weeks previous to a mild attack of lobar pneumonia.

[2] Cadet (quoted by Dr. Ashby, 'Med. Times and Gaz.' March 25, 1882) recognises pneumonia in children under three aspects, namely, those with physical signs from the first, those with tardy physical signs, and those without physical signs; the local lesion in these last, as he believes, remaining central.

Collective Investigation Report,' with nearly a seventh part of its total of 1,066 cases (159) referring to children under 12 (a proportion of these, no doubt, being examples of broncho-pneumonia), tells us no more than that with children, as with adults, the disease may depend on insanitary conditions, that it occurs sometimes in epidemic form, and may spread after a manner suggestive of contagion, while in many instances no weather exposure of any kind has preceded its attacks. Some of the causes to which we have alluded as predisposing to pneumonia in the adult, such as acute alcoholism, obviously have little place with children, and it must be admitted that a large number of these patients suffer the disease from no discoverable cause. Yet there is evidence to show that children not seldom contract pneumonia after a manner which, if it be not altogether special to childhood, is, at the least, more common and more obvious at that period. We have already mentioned the sudden appearance of pneumonia, the sequent of a long train of symptoms, some nervous, some intestinal or gastric, which have endured for weeks, and which thereupon subside. Very striking sometimes is the occurrence of the disease, quite apart from exposure, after an unwholesome meal of which the immediate consequence is vomiting and purging, to which the lung inflammation succeeds. In such cases, without framing any exact hypothesis as to the nature of the connection, it seems obvious from the facts that *gastro-intestinal disturbance*, so easily excited in infancy, is the first in a chain of events that sooner or later issue in pneumonia.

The *mortality* of pneumonia in children is certainly very small. It is true that young children die not infrequently with symptoms indistinguishable from that disease during life. But, for reasons that have been given, the diagnosis needs to be verified post mortem. What we know is that the hepatised lymph-covered lung of primary pneumonia is but rarely seen in dead children. We have put this to proof in the following calculation from the Records of the Hospital for Sick Children. During a period of five years out of a total of 258 children examined after death (July 1870–5), there occur 43 examples of well-marked lung consolidation, variously disposed and described as pneumonic, cases of broncho-pneumonia being as far as possible excluded. Of these 43 three only represent acute pneumonia, and only one of these is primary. The 40 are examples of secondary lung inflammation as follows:—7 are hypostatic consolidation in connection with prolonged disease, where without active symptoms the patients have gradually wasted to death; 5 are consecutive upon heart disease; 15 are in connection with scarlatinal dropsy, enteric fever, measles, and diphtheria, one of these, following typhoid, being possibly true pneumonia; 7 are cases of rickets where the lungs are carnified and in parts collapsed, the subjects much emaciated, dying slowly of bronchitis, with much dyspnœa, but little fever (in one of these temperature was 99·8, while the respirations were 74); 2 were tubercular; 1 was consolidated lung surrounding a gangrenous cavity; 3 were cases of consolidation following pericarditis.[1]

Turning now to the three examples which represent the mortality of lobar pneumonia in children during this period of five years, it appears that two of these are associated with pericarditis, while the third is a case of double pneumonia in a child of two years and a half, being the sole example of simple acute pneumonia in the whole series. The following abstract of these three cases may be here inserted :

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[1] These three alone of the forty have any near resemblance to pneumonia in pyrexia, duration and morbid anatomy. Two of them had recent pleurisy, and the consolidation affected one lung only. In the third the lungs were bound down by old adhesions. The youngest of the three was two years and four months old. The upper lobe of the right lung was here consolidated, the middle lobe in part collapsed, and the lowest tough and carnified; the pleura having recent lymph. The pericardium contained eight ounces of purulent fluid and some lymph. We have thus the essential symptoms of pneumonia, but not idiopathic, inasmuch as it followed pericarditis, a sequence commoner with children than with adults.

The first is that of a well-nourished, healthy child, nine years old, who died after six days' illness, with dyspnœa for the last three days only. The earliest symptom was nausea with vomiting, and soon a hacking cough. On admission, two days before death, dyspnœa was urgent, with orthopnœa and much restlessness, the face being cyanotic. No pericardial rubbing was to be heard, and the physical signs indicated consolidation of the left lung. The condition of restlessness and dyspnœa continued up to the child's death. On post-mortem examination both lungs were found covered with opaque corpuscular lymph, ¼ inch or more in thickness. The external surface of the pericardium was also covered with lymph, and within it was serum, with lymph shaggy and villous. The left lung as to its lower lobe was solid and red, with signs of grey mottling in one or two places. The upper lobe of this lung and the whole of the right lung were greatly engorged, and the lower half of the inferior lobe of the right was collapsed, and sank in water. The left lung weighed 11½ oz., the right 9¼ oz.

The second case was that of a girl of ten. Chorea of three weeks' standing had been followed by pericardial rubbing and signs of pleurisy of the left side. The child died suddenly, with blue face and jactitation, as one asphyxiated. On inspection the pericardium was found adherent by recent lymph, as was also the left lung. Some fibrin coated the aortic and mitral valves. There was no absolute solidity on the part of the lungs, but both were congested and œdematous, and there was pleurisy in connection with the left.

The third case is described as double pneumonia, but is somewhat imperfect. The lower lobe of the right lung was 'solid, firm, and somewhat tough ;' there was some recent lymph on the surface of the left. The child (aged two and a-half) was ill twelve days in all ; it was 'at times short of breath ;' P. 136, R. 48. Some time before death it became unconscious.

Strictly speaking, then, there is but one case of simple pneumonia, the other two being preceded by pericarditis, an association which does not appear greatly to modify the phenomena of the disease, though it may suffice to render it fatal.[1]

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[1] Similar conclusions, in reference to the small mortality of child's pneumonia, are recorded by many observers. Dr. Townsend of Boston

While it is impossible, as we have shown, to make a complete separation between lobar and *broncho pneumonia* in children, nevertheless the contrast between them is sufficiently striking in characteristic examples, and it concerns equally the causes, symptoms, duration, and sequelæ of the two affections.

Thus in broncho-pneumonia (1) the subjects are infants rather than young children, especially the ricketty and syphilitic and those improperly or insufficiently fed, as well as sufferers from certain specific fevers, mainly measles and whooping cough.

(2) The antecedent disease is bronchitis, upon which the alveolar inflammation is engrafted partly, as some maintain, through the intervention of collapse. This extension of bronchitis to the pulmonary alveoli is so common in infants, that it is vain to seek for any special exciting cause other than the bronchial inflammation of which it is the sequel, although no doubt exposure, neglect, and unsuitable food, render such extension the more probable.

(3) The affection is always bilateral, yet it may be so unequal in its distribution as to appear one-sided, and so arranged as regards proximity of the inflamed areas as to be indistinguishable physically during life from lobar pneumonia.

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gives statistics of great interest upon this subject, whence it appears that, out of 1,138 cases gathered from various sources, the mortality of primary pneumonia is 28, or a little over 2 per cent. The largest numbers quoted in this list are the following :

Barthez	212 with 2 fatal
The Pendlebury Hospital . .	234 ,, 3 ,,
Ziemssen	201 ,, 7 ,,

Dr. Townsend does not include the 'Collective Investigation Report,' believing, rightly as we think, that no sufficient distinction is there made between lobar and broncho-pneumonia, and he adds, with reason, 'unfortunately lobar and broncho-pneumonia are so frequently classed together that the idea prevails that pneumonia in children is a very serious and fatal disease.'

See Townsend on Acute Lobar Pneumonia in Children, 'Archives of Pediatrics,' March and April 1889.

(4) Its course and duration are extremely variable, so that no temporary amendment gives promise of near recovery, and no average taken from a number of cases would supply any notion as to the duration to be expected in a given case.

(5) Its temperature is erratic, and the tracing of to-day gives no forecast as to to-morrow. A heightened temperature may concur with general improvement, and a lowered, or very low temperature, with general decline. Relapses and recurrent attacks are very frequent, and (as with bronchitis itself) a few repetitions suffice to establish a morbid liability thus to suffer as well as a predisposition to tubercle.

(6) The best measure of the child's condition from day to day is not the temperature nor the physical signs—these may be absolutely delusive—it is the pulse, the greater or less stress of attending symptoms, such as vomiting, diarrhœa, and wasting; and, more than all, it is the rate of breathing, the degree in which the ribs, intercostal spaces, and epigastrium are sucked in with inspiration, the kind of cough, whether strong or feeble or disappearing altogether from mere debility. In the early stage the differential diagnosis of broncho-pneumonia may be difficult or impossible,[1] and, except for certain general rules of guidance, which it is easy to press too far, prognosis is in much the same position.

If this description be compared with that of lobar pneumonia in the sudden origin, definite course and permanent recovery of the latter, it will be seen how wide is the difference between these two forms of lung inflammation, notwithstanding their anatomical kinship and the fact just mentioned that in particular instances they may be indistinguishable.

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[1] It is pointed out by Henoch (p. 385 *loc. cit.*) that severe dyspnœa may arise in children without secretion or definite physical signs, owing to hyperæmic swelling of the mucous membrane, and consequent narrowing of the lumen of the smaller bronchi.

Of the *sequelæ* of acute pneumonia in children all that need be added to what is stated in Chapter VI has reference (1) to the occurrence with much greater frequency than in adults of a chronic condition, due to the retention in the lung of the material products of inflammation ; and (2) to the occurrence of empyema.

Chronic pneumonia, although not irrecoverable or necessarily spoiling to the lung tissue, is, from the character of the physical signs, very apt to be mistaken for phthisis. In such cases, while recovery from the acute attack seems to be complete, judging from the general health and increase of weight, the early signs of consolidation remain absolutely unaltered. Yet it is certain that a proportion of these children recover, and the event is sufficiently common to justify special caution before attributing pulmonary phthisis to children upon physical evidence only.

The following example is of a pattern with many others, but in hospital practice the patients are so often lost sight of that it would be hazardous to estimate the proportion that fully recover.

Sophia T., six years and a half old, was admitted to the Hospital for Sick Children, November 27, 1873. Her uncle had died of phthisis. She had been ill a month with cough and pain in the chest, spitting thick, uncoloured phlegm. Latterly the symptoms had increased, and some diarrhœa had been noticed. The child was now acutely febrile, having a pulse of 182, and temperature 104·8. The whole of the left side posteriorly was dull to percussion with increased vocal fremitus, and over the lower part, but most markedly immediately below the scapular spine, respiration was intensely tubular. There were moist crackling sounds over both lungs. The notes of progress, extending as they do over several months, must be here summarised. It may suffice to say that such progress was very uneven and erratic, the temperature rising and falling irregularly, while the child's appetite, weight, and general condition were not less variable. She had profuse sweating at times, the sputa were muco-purulent, with sometimes a little blood. These changes in the child's health corresponded with intercurrent attacks of bronchitis to which she was liable. The lung remaining solid throughout the whole eleven months of observation, was yet free from time to time of all added sound, and in such intervals fever would disappear, the appetite become voracious, and the child rapidly gain in flesh, until, with renewed bronchitis, temperature would again rise, and the general health decline. Upon measurement it appeared there was some contraction of the left or affected side in all its diameters. The variations in the child's weight indicate in some degree the course of the complaint. On admission in November she weighed 2 stone 3½ pounds; this weight increased with slight oscillations to 2 stone 7 pounds in May of the following year, the date of her transfer to convalescent home.

Of the further progress little need be said. The bronchial attacks were still frequent, but caused less general disturbance than formerly; there was neither hæmoptysis nor any form of spitting. As regards physical signs, while the tubular breathing remained, the dulness of the left thorax diminished in extent and degree, and from the alteration in voice sound and vocal fremitus which occurred, the area of consolidation appeared to be less, and probably now confined to the central portion of the lung. So the child left the hospital, after a residence of eleven months, apparently well, but with the material results of pneumonia still present

Of the other not rare sequel of child's pneumonia, viz. *empyema*, many illustrations could be given. But to insert these would transgress the proper limits of our subject. It may suffice to say that at this time of life empyema is both more frequent and more insidious than with adults. Young children, therefore, when convalescent after pneumonia, must be carefully watched and physically examined from time to time, with a view to this complication.[1]

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[1] These pus collections may be found sometimes post-mortem in an early stage, and as yet too scanty to afford destinctive physical signs. Thus a child of eighteen months under our care at the Hospital for Sick Children for lobar pneumonia of which it recovered, but shortly to die with bronchitis and purulent pericarditis, presented the following appearance post-mortem as regards the right upper lobe: 'The pleural surface of the lung is covered with a thick layer of yellowish buttery lymph lying between the

The subject of *Treatment* will be discussed later, and needs but brief notice here in reference to the special subject of the present chapter. Its principles and many of its details are the same, whatever the age of the patient, and what is said now must be regarded as merely supplementary to that which is to follow presently referring to treatment in general. Yet for convenience this place may be chosen for some remarks upon certain features of pneumonia in infancy and childhood demanding special measures such as do not apply in later life. These concern (1) feeding; (2) local applications for relief of pain and insuring equable temperature; (3) remedies for certain initial symptoms, such as diarrhœa and vomiting, far commoner with children than adults; and in respect of symptoms of special danger—symptoms precisely the same as occur in later life; (4) means of stimulation applicable to children; and (5) the best method of blood-letting in these subjects.

We have spoken of lobar pneumonia as rarely fatal to children, as well as of the uncertainty of prognosis in the case of broncho-pneumonia. In practice, however, as has been shown, the two forms are not always distinguishable, and in the matter of treatment nothing is gained by separating them so long as it is remembered that the outlook is more or less favourable, *cæteris paribus*, according as the previous history and the physical signs show less or more of bronchitis.

In all acute pulmonary diseases of infants at the breast, the first care of the physician concerns *feeding*. It is apt to be forgotten that with urgent dyspnœa, and sometimes mucous obstruction of the nasal passages as well, the child is no longer able to suck efficiently, and that the effort to do so, if continued, exhausts strength to little purpose. Milk or cream in

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layers of pleura and just sufficient to separate them. This condition is confined to the upper lobe, and is apparently a sequel to pneumonia of this lobe.'—Notes by Dr. Voelcker, 'Post-Mortem and Case Book,' vol. viii. p. 400.

barley water, with a few drops of saccharine solution of lime in proportions suitable to the age of the child, will have to be substituted for the mother's milk, although this latter need not be wholly withheld, if given in a teaspoon or partially by suckling, the effect being carefully watched.

The frequency of diarrhœa and vomiting at the commencement of the attack calls for special precaution in respect of diet with babies and young children. Where cow's milk cannot be digested, or where it seems to provoke diarrhœa, peptonised condensed milk may be substituted, or diluted raw meat juice [1] with cream. If cow's milk be given, it should be boiled so long as diarrhœa persists.

Beef-tea, even when well made, the meat being soaked for an hour in cold water, affords a poor food for young children, and, like raw cow's milk, is apt to cause diarrhœa. It contains, as Dr. Cheadle has shown, a low percentage of proteid, especially when compared with raw meat juice. Yet it must be added that well-made beef-tea is superior in this respect to some of the 'patent' essences and juices of the shops, and with the elder children serves very well.[2]

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[1] Raw meat juice prepared after the following manner is of high nutritive value, and far better for children, in Dr. Cheadle's opinion, to that he obtained by Liebig's process with the addition of hydrochloric acid (see Chap. XX). It has been largely employed of late at the Hospital for Sick Children.

Mince finely the best raw rumpsteak ; add cold water, one part to four of the meat ; stir well and allow to soak for half an hour cold. Forcibly express juice through muslin.

ANALYSIS AND COMPARISON WITH BEEF-TEA

Raw Meat Juice

Proteid (albumen)	5·1	Total Nitrogenous Elements 8·2
Extractive	3·1	
Salts	0·7	

Best Beef-tea (1 *lb. meat to* 1 *pint*)

Proteid (albumen)	1.02	Total Nitrogenous Elements 2·84
Extractive	1·82	
Salts	·88	

[2] The subject of artificial foods and the physiological facts to be kept

With children, even more than with adults, the even temperature of the sick room, its ventilation, and, when cough is irritable, the sufficient moisture of its atmosphere, are points of treatment of the highest importance. Cotton wool, for its lightness and convenience of removal, is the most suitable material for swathing the chest ; but where dyspnœa is severe and there is any appearance of pallor or blueness about the face, the substitution for a few minutes of a flannel heated in the oven is of service. It is too common to find infants overdone with useless wrappings. Observation of the patient's catching respirations with restlessness and moaning will indicate pleuritic stitch, and this, which is seldom a symptom of long duration, may be removed or mitigated by hot poultices of linseed and mustard, or sometimes by ice (Chap. XXI), while pyrexia, if reaching 104°, may be reduced by tepid sponging.

With this much of treatment, judiciously carried out, and without drugs, we believe that young children with pneumonia and broncho-pneumonia, in a majority of cases, fare as well as when various expectorants, stimulants, and supposed sedatives are given.

Medicines, and especially ipecacuanha and squills, are often administered, in our belief, without real need and as a mere routine, with the effect of producing nausea and preventing that sufficient nourishment which is the cardinal matter for consideration. Yet inasmuch as diarrhœa, as we have seen, is not rarely among the initial symptoms, more than one indication may often be met by the cautious use of opium, mindful always of the easy narcotism of children. Thus a few drops of paregoric, suitable to the age, may be combined with spirit of chloroform and given with syrup, with the effect of quieting

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in view in regard to farinaceous diet for young children is discussed in much detail by Dr. Cheadle. See 'Artificial Feeding and Food Disorders of Infants,' p. 95 *et seq.*

both cough and diarrhœa. Yet it must be remembered (1) that both these symptoms tend to disappear in lobar pneumonia as the disease develops, and (2) that when the characteristic drowsiness is pronounced or there is much delirium, all forms of opium must be given with great caution. With both lobar and lobular pneumonia, our practice is to wait and watch the development of the disease—watching, above all, the rate and manner of respiration from hour to hour—with as little meddling as possible, until or unless some urgent symptoms call for further treatment.

Of such urgent symptoms, the most important and the gravest is *extreme dyspnœa.* In young children, shallow ineffectual respirations may exceed 100 per minute. But such rate of breathing cannot be maintained for any length of time without the lungs becoming engorged, and probably in part collapsed. The condition, therefore, is to be regarded as tending towards death, and it must be combated without delay. It is, undoubtedly, true that a large proportion of children who come to this pass die; and bleeding, like every other expedient used only in extremity, gets disparaged accordingly; it is true also that extreme dyspnœa in broncho-pneumonia, like all its symptoms, will sometimes cease of itself. But in our belief the treatment that most promotes recovery in this emergency is the free abstraction of blood, and the best method of bleeding with children is by leeches applied to the chest, the application to be followed, should pyrexia exceed 103·5, by the wet pack or tepid sponging, and in any case stimulants being freely given. In cases of the kind we have repeatedly failed to obtain sufficient blood flow from bleeding in the arm.[1]

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[1] See 'Two Cases of Broncho-pneumonia treated with Bleeding and Ice,' by Dr. David B. Lees, 'Brit. Med. Journ.' July 11, 1885; 'Treatment of Pneumonia by the Ice-bag,' by the same, 'Brit. Med. Journ.' October 26, 1889. The subject of the application of ice will be discussed more fully in the chapter devoted to Treatment. As regards bleeding, a case recently

In the case of *extreme prostration* less is to be gained by the use of alcohol than with adults; it must be used with more caution and not continuously. To infants not more than a few minims of brandy can be safely given for a dose, to be repeated at short intervals over a limited time, watching its effect by the index of respirations, pulse, and state of consciousness after each administration. The drowsiness so characteristic of the affection in young children warns us to dispense with it as far as possible.

Delirium which, as we have said, is not uncommon with the earlier symptoms, needs careful and constant watching, so that the child may be soothed from time to time by the hand and voice of an attendant. When it concurs with high fever, occasional tepid sponging will often relieve both symptoms to a remarkable degree, and enable the patient to sleep.

Cold and tepid baths are unsuited to children, owing to

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under our own treatment may here be quoted. 'Daisy B., a baby of three months, was admitted to the Westminster Hospital, September 8, 1889, for cough and shortness of breath of four days' duration, but which had become suddenly much worse the day before. Temperature was 104°, pulse 186, and respiration (counted repeatedly) 108; the face livid. Physical examination elicited dulness, fine crepitation and increased vocal fremitus at the base of the right, and signs of consolidation less marked at the apex of the left lung. Three leeches were applied to the anterior chest, and the child was ordered three minims of brandy every half hour, and a mixture of spirit of chloroform. The following is the note of the house physician (Mr. Haig): The leeches were applied soon after the visit, the lividity and distress being very marked. Bleeding continued some time after the leeches were removed, and was eventually stopped by application of perchloride of iron. The condition of the child at the moment looked very critical, and brandy had to be given freely. The improvement in respiration, however, was very marked, and the child was soon quietly sleeping. The following day temperature did not rise above 100°, pulse was 142, and respirations 63. The day succeeding the child was practically convalescent, and it was discharged on the 17th.'

A second case might be quoted in an older child very similar to this one, in that the amount of blood lost was much more than was intended, owing to hæmorrhage continuing after the leeches were removed, the patient becoming pale and faint, but, as with the baby, alarming dyspnœa was relieved in the same striking way. It need not be added that we have cases to quote where bleeding has *not* thus relieved dyspnœa nor saved the child.

their depressing action on the heart. As with quinine and other reputed antipyretics the reduction of temperature is brief, and the treatment needs constant repetition. But for wet compresses applied to the chest there is much to be said, especially in broncho-pneumonia. A napkin or towel dipped in water is wrung out and applied lightly round the chest, some cotton wool being wrapped round it. The application is changed more or less often according to the child's temperature and number of respirations. The chief use of this form of compress seems to consist in the deep inspiration it occasions on every fresh application.[1]

As regards *broncho-pneumonia* much difference of opinion prevails as to the need or the use of active drug treatment. We agree with Dr. Eustace Smith and others in believing that medication is of very secondary importance. In suitable cases —that is to say, where prostration is not marked and there is physical evidence of much secretion within the lungs—we have found a full emetic of ipecacuanha useful in removing the mucous collection and relieving dyspnœa. But we doubt whether this remedy is of more than temporary service, and considering the feeble constitution of most children suffering from the disease, it will seldom bear repetition. Counter-irritation with mustard and linseed will sometimes give manifest comfort to the child, and be followed by deeper and slower respirations. But for extreme and dangerous dyspnœa there is, in our belief, no such remedy as the withdrawal of blood on precisely the same plan as with lobar pneumonia. Leeching answers the purpose well, and certainly better than the lancet. Of dry cupping commended by Prof. Henoch Eustace Smith, and others, we have less experience. Just as in lobar pneumonia the advent of crisis often leaves us in doubt as to whether nature or the supposed remedy is to be thanked for sudden improvement, so in the bronchial form, the frequent and rapid transitions of the disease make it impossible to determine accurately the effect of treatment. Yet in both cases alike there are certain emergencies, of which the danger increases with the duration, and which cannot be suffered to continue without some attempt on our part at alleviation. We use those means that, as a matter of personal experience, have most often been followed by improvement; but the changeful character of the disease, its extremely doubtful prognosis, and the failure from time to time of all remedies alike, combine to make it irrational to commend, without reservation, any method of treatment whatever. It need not be repeated that treatment reserved for desperate straits must inevitably be associated in the mind with repeated failure, and is not to be condemned on that account.

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[1] Henoch, vol. i. pp. 397, 414, *et seq.*, New Sydenham Society.

The treatment of the sequelæ of pneumonia is beyond the scope of this chapter. It will suffice to remark here in reference to the most important of these, empyema, that the modern plan of free drainage and rib resection has rendered its prognosis very much more favourable than heretofore, and that the large majority of such cases are now cured.[1]

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[1] See 'Forms of Empyema in young Children and their Treatment,' 'Westminster Hospital Reports,' vol. v.

CHAPTER XIII

EPIDEMICS

Prevalence of epidemics in past times—The Iceland epidemic of 1863—Records of village epidemics—The Middlesbrough outbreak, 1888—Outbreaks in prisons, reformatories, &c.—The question of contagion—Influenza as a precursor.

We have already briefly alluded to the fact that most of our knowledge of pneumonia in early times is derived from accounts of 'epidemics,' some of great extent and severity. At this distance of time, and with the necessarily imperfect description of morbid characters that has been transmitted to us, it is not possible to accurately discriminate in these records the instances of genuine pneumonia, as we now know it, from cases where the pulmonary conditions formed but part of a general infective disease. It is conceivable that some of these outbreaks were really examples of typhus, or typhoid fevers, or of influenza, and therefore did not strictly belong to primary pneumonia. Nevertheless, the records of modern days, since the introduction of auscultation, leave no doubt as to the comparatively frequent occurrence of pneumonia in epidemic form, and, in some striking instances, with evidence of infection or contagious transmission. Hence, it behoves us to devote some space to the consideration of this branch of the subject, which, as will be seen, is of great etiological importance. It has, moreover, received additional interest since bacteriology has gone far to establish the association of pneumonia with definite forms of micro-organisms.

It would hardly be possible to review the whole literature of epidemic pneumonia within our present limits. It has attained vast proportions, and has several times been most carefully analysed.[1] All that we can do is to make a few selections from literature as examples of various types of such epidemics,[2] and, following the example of Mendelsohn,[3] we may divide the instances into groups, according to the extent over which the disease prevailed. Thus there are records of epidemics occurring over large areas of country or throughout large towns and districts; others where small villages, or even a few streets in a town, were alone attacked; others where the outbreak occurred within public buildings, as barracks, prisons, reformatories, and schools; until finally we come to groups of cases arising within the narrow area of one family or dwelling, and sometimes under circumstances where it is almost impossible to avoid the conclusion of a contagious influence.

It is obvious that the conditions under which the disease arises in these several circumstances must be widely different. The larger the area over which it prevails, the greater the probability of its being mainly due to some unusual prevailing condition, meteorological or other;[4] whilst, in the more restricted ‘epidemics,’ there is likely to be evidence of defective sanitation, of conditions termed ‘pythogenic,’ and in some cases even of actual contagion. This diversity in the manner of occurrence and in the extent of epidemics of pneumonia, renders it more difficult to accept the doctrine of 'pneumonic fever' in its entirety—or at least to rely for its proof upon the existence of epidemic prevalence, apart from other considerations.

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[1] See Hirsch, ‘Handbook of Geographical and Historical Pathology,’ 2nd ed. vol. iii. (‘Syd. Soc. Trans.’), 1886; Haeser, ‘Lehrb. d. Geschich. d. Med.’ 3rd ed. 1882, vol. iii.; Sanders, in ‘Seguin’s Archives,’ 1881, June and August; Mendelsohn, ‘Zeitschr. f. klin. Med.’ 1883; Wells, ‘Journ. Amer. Med. Assoc.’ 1889, February 23, where most ample references will be found.

[2] The summary given by us in the ‘Collective Investigation Record,’ vol. ii. 1884, will be largely drawn upon for this purpose.

[3] *Loc. cit.* p. 178.

[4] As in the case of the ‘Alpenstich.’

One of the most striking epidemics of this disease is that which occurred in Iceland in the year 1863, and is reported by Dr. Hjaltelin,[1] inspecting medical officer, who, although he had practised in that country for ten years, had not previously met with pneumonia save in the sporadic form. The outbreak had been preceded by an epidemic of influenza, and it prevailed to its largest extent in January and February, a period of severe storms, and a dry, cold air impregnated with ozone, to which he ascribes the high mortality from respiratory disease in northern latitudes, and the frequency of pneumonia amongst the Eskimos. We may quote a few passages from Dr. Hjaltelin's report.

'The winter,' says he, 'was an extremely stormy and rough one in this island. People were still in many places, and especially in the north, newly recovered from the epidemic dominion of the influenza, and it seemed to me quite natural that their respiratory organs were still in a state of great sensibility, which might give occasion to acute pulmonary affections during that extremely rough and stormy winter season.' The author, whose vigorous narrative seems to suit the high latitude from which it comes, proceeds to detail the leading features of eighty cases, of which eight died, four of them being past hope when first seen. In two of these death took place after four days' illness, one of them having 'walked about' the day before the seizure. In both cases extensive hepatisation, lymph-covered pleura, coagula in the right ventricle, and granular section, indicate beyond doubt genuine pneumonia, fatal at an early stage.

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[1] 'Edin. Med. Journ.' April 1864.

After speaking highly, from personal observation, of the results obtained in Germany by the 'water doctors,' the writer goes on to relate how, having been inclined to no light degree of scepticism regarding the usefulness of blood-letting in pneumonia, his experience of this particular epidemic impelled him, nevertheless, to the practice of it. 'Many cases,' he says, 'convinced me of its necessity in the severer cases, and these were far more numerous than I formerly would have expected.' 'There is no doubt,' he adds, 'that it may be a rare occurrence in the warmer climates of Europe to see healthy and strong people so rapidly affected by a pulmonary inflammation that death follows in two or three days; but this happened very often in our epidemic, and was the general rule where the disease was left alone. I saw several, even young, strong, and healthy people, who the day before the attack were at their work without any complaint, but who next day were nearly breathless, with all the symptoms of the most acute pleuro-pneumonia. I know a parish in the interior of our country with only three hundred souls, where nineteen persons died thus affected in a very short time. Having been more than sixteen years in foreign countries, I never saw any case of pneumonia which could be compared with the common cases of this our epidemic.'[1]

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[1] 'It would no doubt,' writes this Icelandic physician, 'have been a good lesson for the medical men of the nothing-doing treatment to see the ravages of this frightful disease amongst the helpless inhabitants in all the parishes where no medical aid was to be had, and they would have been able to see that their healing process of nature, which they call the natural progress, is, in a really acute and malignant occurrence of this disease, really death, and nothing but death. *Nearly all the patients who were without any medical aid died in three to six days.* Thus in the parish of Gardar, with about 1,200 souls and where medical aid seldom could be had, out of twenty-four patients seventeen died. In the parish of Utskaler, with 900 souls, where nineteen fell sick, and where medical aid could not be obtained, eleven died. In the parish of Kalfatjorn, with 800,

Coming thus quite unwillingly to the practice of phlebotomy, it is the more interesting to notice the result obtained by its means :—'The insupportable pain beneath the right nipple across the chest, the great dyspnœa which in some seemed to threaten with immediate danger, the disturbed balance in the circulation, were generally greatly relieved ; and the hard, incompressible, but often irregular pulse became more regular after the loss of eight or twelve ounces of blood. The relief thus obtained was not always momentary, but would last for several hours or even one day ; and although the rapid and dangerous pulmonary congestion could by no means be subdued by this remedy, it was evidently of great service to calm the storm.'

These observations from Scandinavia, referring, as they do, to the prevalence of an undoubted pneumonia, whose natural tendency was not towards recovery but towards death, are of unique value to us now as exhibiting—if we may so say—the possibilities of the disease. What has happened to the Icelanders may happen to us. In restricting our view to a particular time and country, we do less than justice to the practice of foreigners and of our predecessors, and are far too complacent in regard to our own.

An epidemic of pneumonia, in many respects similar to that described by Dr. Hjaltelin, occurred to the 22nd Regiment at New Brunswick. It is fully narrated by Dr. Welsh in the 'Army Medical Reports' for 1867, and was remarkable in two respects, first, that in the coldest month of the three over which the epidemic spread, the admissions into hospital from pneumonia were much fewer than for the following and warmer month ; and, secondly, that that portion only of the regiment suffered which was quartered in a new building abounding in

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souls, nearly all the affected, this number being 14, died.' So it was everywhere, without medical interference.

draughts. Among the women and children, who were better housed, only two cases of pneumonia occurred.

In contrast with such narratives we have accounts of epidemics, both ancient and modern, which, while they are described as pneumonia, differ very widely from the description just given in their origin, course, and response to treatment. We have spoken already of such sicknesses in olden time; of the implication of the lungs, not by hepatisation, but in a general congestion tending rapidly to gangrene or what is called 'purulent destruction'; of the coincident liquid effusion in place of pleural lymph; of the associated dysentery or ulceration or diarrhœa. The medical history of our own day will supply similar illustrations. Thus, Dr. Bryson has preserved an account of an epidemic pleuro-pneumonia, in some ships of the Mediterranean Fleet, in 1860.[1] The disease was of the 'asthenic or typhoid type'; there was great congestion of the lungs, and in the ship that suffered most (owing, as appeared, to excessive overcrowding) scorbutic symptoms arose. Effusion into the chest was discovered in a few instances. Diarrhœa and dysentery were common. It was suspected that the sick landed at Malta communicated the affection to the patients amongst whom they were placed. There are further narratives, both at home and abroad, to a similar effect. In Ireland the mortality ascribed to pneumonia was largely increased during the years of famine, when dysentery, typhus, and scurvy were also common. Ziemssen[2] believes that the variations of pneumonia are in proportion to the density of the population, that the mortality of typhus and of pneumonia are in close correspondence, and that the two diseases are due to similar causes.

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[1] 'Lancet,' Jan. 9, 1864.

[2] 'Edinburgh Medical Journal,' vol. iv. p. 380. Unfortunately, in dealing with large numbers and trusting to tables of mortality, the conclusions attainable are of a very general kind. From the concurrence of testimony it is made clear that *some form or other* of lung-inflammation is favoured by the causes alleged, but, except for Dr. Bryson's account, the precise condition of the organ in individual cases is not described.

In the winter and spring of 1877–8, an epidemic of so-called 'typhoid' pneumonia occurred in Florence and other parts of Tuscany,[1] during the prevalence of typhoid fever. The disease attacked individuals of all classes and ages, and appears to have been of a specially malignant type, being notable for its asthenic character, and, when recovery took place (which was seldom), in its termination by lysis. In some cases the onset was preceded by bronchial catarrh ; in others it was abrupt, but there was mostly a prodromal period of malaise, headache and prostration, whilst during the course of the fever the physical signs of pulmonary inflammation were often absent ; in some cases their appearance was delayed until the seventh or eighth day ; in some, the indications were those of consolidation spreading through the lung. Many cases were examined by Banti after death, and in addition to pneumonic hepatisation and hæmorrhagic congestion, the chief conditions found were a dark colour and fluidity of the blood, fatty degeneration of the heart muscle, and softening of the spleen ; but in no case were the intestinal lesions of typhoid fever present.

There are several records of village epidemics in literature, and in some of them there is rather strong evidence in favour of the infective or even the contagious character of the disease. This at any rate is the case with the epidemic which occurred in 1881, at Becherbach, recorded by Butry :[2] During a severe winter, with N.E. winds, there had been only a few cases of pneumonia, and these of a mild type ; but in the spring a malignant outbreak occurred. In a population of 400, no fewer than 20 persons were attacked within a few weeks, in April and May ; and 10 (*i.e.* 50 per cent.) died. The disease spread mainly amongst relatives and neighbours, and those who visited the sick at their homes. It was of the asthenic type, and in seven of the cases the upper lobe was involved. There was no splenic swelling and jaundice occurred in several of the fatal cases. In those that recovered the convalescence was protracted.

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[1] Banti, 'Arch. Gen. de Med.' 1880, ii. p. 36.

[2] 'Deutsch. Arch. f. klin. Med.' 1881, xxix. p. 193.

In the same year (1881) and season, an epidemic took place at Rietnordhausen,[1] a village of 700 inhabitants, seven kilometres from Artern. It was almost entirely confined to children —all but three of the 42 attacked being below the age of 12 years. The etiology of this outbreak seems to have been clearly established,—the village school-house, at the foot of the hill on which the village stands, being situated near to a graveyard and stagnant pond. It is suggested that the N.E. wind, which set in a few days before the first child sickened, must have conveyed the emanations from the pond directly across the graveyard to the school buildings.

Lustnau (1633 inhabitants), near Tübingen, was, from January to May 1881, the seat of an epidemic[2]—the first 29 cases arising within a period of 56 days, after there was an interval of 14 days, until March 12, when a recurrence of the epidemic took place. The majority of those attacked were adults, and 60 per cent. were males. The disease broke out simultaneously in many houses, and spread over the whole village, prevailing more in some parts than in others ; and in some houses more than one case occurred. Thirty cases happened in houses which had in previous years contained patients with pneumonia. The rainfall during the period over which the epidemic lasted was comparatively small, and the winter had been the mildest since 1874. The mortality rate was high, ten deaths, or 23 per cent.

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[1] Penkert, 'Berl. klin. Woch.' 1881, Nos. 40 and 41.

[2] Fully recorded by Scheef, 'Croupöse Pneumonie,' herausgegeb. v. Th. Jürgensen, Tübingen, 1883, p. 97.

In all the fatal cases (full details of which are given) there was pleuritis, in some purulent; in many, pericarditis, and in many subserous and submucous hæmorrhages; also parenchymatous degeneration of the liver, spleen and kidneys. In no single instance could exposure to cold be adduced as the cause of the illness; and in many cases the attack was preceded by prodomal symptoms. Ten of the cases terminated by *lysis.*[1]

Another well-marked epidemic occurred at Erbenheim,[2] generally a healthy village (1,500 inhabitants), at a moderately high situation. Pneumonia—which as a rule did not amount to 4 per cent. of all diseases—prevailed to a remarkable degree in November 1882, when 59 cases arose within an area of three miles, the great majority being children. There were only five deaths, or 2·95 per cent. In most cases a crisis occurred from the third to eighth day, and the chief complication was diarrhœa (half of the cases). The disease seemed to select particular streets, and especially neighbouring houses. In ten families two cases occurred, and in six families there were three cases in each.

An interesting village epidemic is recorded by Roudet.[3] The place was Plairieu-s.-Saduc, and the first to be attacked was a communal guard, who lodged in a house where there was a school. He fell ill on March 28, and recovered, but on April 9, one of the schoolgirls, aged eight, developed pneumonia. She was well in a week, and on April 13, her sister, aged fifteen, was attacked with pneumonia. On the 14th, their grandmother, who had nursed these children, was attacked, and died on the

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[1] Abstract in 'Coll. Investig. Rec.' vol. ii. p. 17.

[2] Senfft, 'Berl. klin. Woch.' 1883, No. 38. For other records see Moellmann, 'Berl. klin. Woch.' 1879, No. 14; Feldhausen, 'Schmidt's Jahrb.' 1882, p. 256; v. Holwede and Münnich, 'Berl. klin. Woch.' 1881, p. 332; Schmid, 'Berl. klin. Woch.' 1883, p. 346. Abstracts of these observations are given in the Report of the Collective Investigation Committee.

[3] 'Soc. Nat. de Méd. de Lyon,' 1887. We quote from a paper by H. Barbier ('Gaz. Méd. de Paris,' June 8, 1889).

19th. On the 21st her brother, who had visited her during her illness, fell ill and died on the 26th. On the 24th, one of her cousins, who had occupied the same room as the children, was also attacked, and succumbed on the 28th. So that within three weeks, there had been five successive cases of pneumonia in one family, three of which were fatal. At the same time other cases occurred in the village, all traceable to the same source. Thus, on the 29th another of the school children and two other persons in the vicinity were attacked. Lastly, the schoolmistress, who occupied the same house as the man first attacked, herself had the disease. Of the nine cases, at least seven were either in one family or had lived in the same house as other patients.

Reference may here be made to the accounts of two epidemics of pneumonia in the Punjaub, described by Surgeon-Major Maunsell.[1] The first of these occurred from July 1882 to March 1883, the largest number of cases being in the month of January. There were 85 cases, and in 39 of these the patient had had ague once during the previous twelve months, but no definite relationship was traced between the pneumonia attack and that fever. The disease, from its complication with bronchitis, and the bilateral implication of the lungs, was deemed to be 'catarrhal' rather than 'croupous pneumonia.' There were five deaths. The second outbreak, almost about the same time as the first, was at Edwardesabad, on the Punjaub frontier of Afghanistan. This was more clearly of the usual 'lobar' type, and many cases were ushered in by gastro-intestinal disturbances.

In this country we may note the unusual prevalence of pneumonia, in the autumn of 1883, at Newcastle-on-Tyne, as related in the report of Mr. Armstrong, Medical Officer of Health. In drawing attention to this, a writer in the *Lancet*

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[1] 'Coll. Investig. Rec.' 1884, vol. ii.

(1884, i. 127) says: 'The facts as regards pneumonia are somewhat striking. It was certainly not due to any special coldness of season, and hence Mr. Armstrong inclines to the view that the disease has probably been of a zymotic character, induced by defective local sanitary circumstances, resembling the specific pneumonia of which much has been written in recent years.'

In Belfast, in the autumn of 1887, a large number of cases of pneumonia occurred, several of which proved fatal; and, at the same time, many cases of sore throat of severe type prevailed. The illness was attributed by many to the prolonged dry season preventing the proper flushing of drains (*Lancet* 1887, Dec. 3, p. 1146).

But perhaps the most remarkable outbreak yet on record is that which occurred in Middlesbrough in 1888, and which has been made the subject of a Local Government Board inquiry.[1] The town, which is filled with an industrial population of nearly 70,000, lost from this disease, from February 25 to July 14, no fewer than 369 of its inhabitants, as many as 288 being males. The clinical features of the attacks were sudden onset, rise of temperature, pain in side, rapid pulse and breathing, usually vomiting or diarrhœa, delirium, accompanied, or soon followed, by the physical signs of pleuro-pneumonia. The expectoration, at first rusty, became prune-juice in colour; and Dr. Klein's investigations[2] proved the presence in the sputa and the hepatised lung of a special form of bacillus. The deaths mostly occurred in from three to five days; and post-mortem examination revealed lobar pneumonia with pleurisy, softening of spleen, and gastric ecchymosis. Dr. Ballard is inclined to regard it as a specific febrile disease, and as being infective. He adopts the term 'pleuro-pneumonic fever' as best describing it; and he found that it was communicated either by direct contact, or through the medium of the sputa, or other excreta of the sick, and also by food. The exposure to 'slag dust,' which had been suggested as a likely cause, was considered by Dr. Ballard to have only a predisposing influence.[1]

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[1] For full details of this epidemic, the reader is referred to the exhaustive and elaborate report by Dr. Ballard ('Report of Medical Officer to Local Government Board—18th Annual Report of the Board—1888–89,' Supplement.) Eyre & Spottiswoode.

[2] Dr. Klein gave an account of his researches in the 'Centralblatt f. Bakteriologie,' bd. v. 19. They are referred to in Chap. XVII, *infra*.

Outbreaks of pneumonia within the comparatively limited area of large institutions, prisons, reformatories, barracks, &c., have been recorded from time to time. In some cases such outbreaks are undoubtedly referable to insanitary conditions, in others there would seem to be clear evidence of contagion.

M. Roudet[2] relates that in the workhouse at Albigny, among 693 inmates there occurred 69 cases of pneumonia from November 10, 1886, to the end of April 1887. At first only males were attacked (13 in number), but after the lapse of thirty-eight days from the commencement of the outbreak the women began to suffer. This was explained by the fact that although the sexes were separate, yet the requirements of the establishment necessitated some communication between the two sides of the house. It is noteworthy that 12 of the bedridden infirmary patients took the disease, and that 5 nurses (2 females, 3 males) also suffered. The same writer gives an account of an outbreak in the two prisons at Lyons (St. Paul and St. Joseph). In May 1886 there were 20 cases among the male prisoners. The epidemic had commenced in February, but it was believed to be traced to the admission in December 1885 of a prisoner who died four days after from the disease, and from a second similar case which developed on January 10, two days after reception. In February another of the prisoners fell ill, and from that time others were successively attacked.

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[1] In view of the foregoing evidence of the undoubted occurrence of pneumonia as an epidemic, it is permissible to suggest that even in large towns, when an unusual prevalence of this disease occurs, there may be the same epidemic influence at work. In the beginning of 1889 there was in some parts of London such a prevalence, for from January 3 to April 10 there were admitted into the Middlesex Hospital 21 cases—an unusually large number—no fewer than 5 of which were fatal. Fifteen were males, and the monthly admissions were: January, 5; February, 5; March, 9 (3 admitted on the 25th, and 3 more on the four succeeding days), and April, 2.

[2] 'Bull. Soc. Nat. de Méd. de Lyons,' 1887; cited by H. Barbier, 'Gaz. Méd. de Paris,' June 8, 1889.

In 1867 there was an epidemic of this nature at the Arkershus prison at Christiania.[1] In the first five months of the year, 62 out of 360 prisoners were attacked with pneumonia, of whom five died. Six nurses were also attacked, and the outbreak was referred to the bad ventilation of the dormitories.

Rodman[2] relates the particulars of an endemic in the Kentucky State Prison, at Frankfort, Ky., in February 1875. Pneumonia had been unusually prevalent in the country during the autumn and winter, but cases had ceased to occur when the remarkable outburst of disease in the prison commenced on February 24. The prison cells, 648 in number, were greatly overcrowded in that month, 694 prisoners being lodged on February 1, and 735 on March 1. During 1874 there had been 75 cases of pneumonia in the prison, with a mortality of 8 per cent.; and from January 1, 1875, to February 24, 16 cases with one death. From February 24 to July 1, there were 118 cases, and 98 of these were of a specially asthenic type, with symptoms pointing to a specific poisoning. Twenty-five died, and of these 14 were new prisoners. The sanitary condition of the prison was very bad, for owing to the uncleanly habits of the inmates, the cells and corridors, especially of the upper floors, reeked with fœtid emanations. The outbreak belongs to the class of 'pythogenic' pneumonia without a doubt; and the high mortality (26 per cent.) justifies the condemnation passed on the prison buildings.

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[1] Dahl, 'Dublin Journ. of Med. Sci.' 1875.

[2] 'Amer. Journ. Med. Sci.' Jan. 1876, p. 76.

Knoevagel[1] has recorded an outbreak which took place at the garrison at Cologne, from October 1, 1879, to May, 1880. During this period, amongst 389 patients admitted into hospital, there were 80 cases of pneumonia, 7 of which proved fatal. Most of the patients came from barracks, which were ill-ventilated and noisome; and, as in other instances, the recruits suffered most.

Grundler[2] published details of an epidemic in the Magdeburg garrison, lasting from September 1873 to June 1874; the number of cases of pneumonia amongst the civil population during that period not being above the normal. The garrison numbered 5,616 men, and of these 73 were taken ill, particularly recruits, and those of an infantry regiment occupying barracks erected on a marshy soil and badly ventilated. Four cases were fatal—*i.e.* 5·48 per cent., so that the outbreak was not a severe one. The winter was mild, but the men were subjected to changes of temperature in their drills, an exposure which may have had some effect in determining the attack.

The most circumstantial account of this class of pneumonia epidemics is that given by Adolf Kühn,[3] as occurring in the Moringer Reformatory in 1874–5. In 1874 there was a great increase in the number of inmates, so that not only was the cubic space of the dormitories much diminished, but beds had to be placed in the corridors. The building was then enlarged to accommodate 237, but the numbers increased to

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[1] 'Schmidt's Jahrb.' 1882, p. 254.

[2] *Ibid.*

[3] 'Deutsch. Arch. f. klin. Med.' 878, xxi. p. 348, and 'Berl. klin. Woch.' 1879, No. 37.

351; and as a consequence there was, during 1874, a great increase in all forms of disease of the respiratory organs, pneumonia and phthisis especially. Amongst them were 11 cases of lobar pneumonia of a peculiar type, and running a course more resembling that of an infectious disease. At the same time there were 43 cases of gastro-intestinal disorder of a typhoid character, associated with bronchitis and lobular pneumonia. At the end of 1874 the buildings were again extended to accommodate 414 inmates, but even this extension failed to keep pace with the number admitted, so that the effects of overcrowding were still felt. During 1875 there were 83 cases of pneumonia, 45 of which were of a severe type. Several of the cases were preceded by malaise, lasting a few days; then occurred a rigor with rise of temperature to 104° or over, but with very scanty physical signs of pneumonia. In such cases there were also swelling of the spleen, albuminuria, and in many diarrhœa. In others the signs of pneumonia were manifest by the third or fourth day, and the disease assumed the 'wandering' type. All the cases were complicated with pleurisy (four with empyema), and in one-fourth of the whole number there was pericarditis. Many had cerebral symptoms. Coincident with these severe and well-marked cases, were others of slight illness, mostly recorded as catarrh, but which from their general similarity were regarded as abortive forms of the graver disease. Post-mortem examinations were made in 16 cases; meningitis was present in 4 out of 7 cases in which the head was examined; pericarditis was found in 10 cases, an enlarged spleen in 13, and some swelling of the intestinal follicular glands in many. Kühn thinks there can be little question of the 'typhoid' character of this epidemic, and that it was apparently due to the overcrowding.

Kerschensteiner[1] describes an epidemic which occurred in the male prison at Amberg, and which might also be attributed to the insanitary condition of the dormitories, but the cases were more strictly of the classical type of pneumonia than those occurring at Moringer. From January 1 to May 28, 1880, 161 inmates were attacked, of whom 46 died, or 28·5 per cent. The epidemic was thus a very fatal one, the cases being of the asthenic type. Kerschensteiner was inclined to regard it as due to some special miasm.

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[1] 'Schmidt's Jahrb.' 1882, p. 254.

It is but a step from such examples as the foregoing to those where the outbreak is practically limited to one house or to the members of one family, of which we shall speak when dealing with the etiological question of contagiousness (Chapter XVI). It may, indeed, be argued that there is no parallel between such limited outbreaks and those which occur on a larger scale. In the former there may be defects in sanitation, and the subjection of a limited number of people to the same noxious influence; in the latter there must be some wider influence at work, although it must be admitted that it has not always been possible to assign any special meteorological conditions sufficient to explain the unusual prevalence of the disease in a particular district.

The relationship between influenza and pneumonia—long noted—and which has been recently brought home to us in the epidemic of 1889-90, has not been satisfactorily explained. Whether the same miasmatic agent, which may be assumed for influenza, is also capable of exciting pneumonia, is one of those questions which must at present remain unsolved. There may be this close connection between the two affections; or, on the other hand, it may be that the remarkably depressing effect of influenza may render its subjects more prone to the action of those agencies which, as we shall show, seem to operate in the production of pneumonia.

CHAPTER XIV

CONGESTION AND OTHER CONDITIONS DISTINGUISHABLE FROM PNEUMONIA

'Congestion of the lungs'—Pulmonary œdema—Hypostatic congestion—Illustrations—Brown induration and its association with cardiac disease—Collapse, lobular and lobar—their differentiation from pneumonic consolidation.

THE term 'congestion of the lungs' is often misapplied. As an active condition, a fluxionary hyperæmia, involving one or both lungs, it is so rare as to be hardly worth considering. Such a condition may conceivably be produced by excessive cardiac action, accompanied with a lowering of arterial tension; or by the inhalation of irritating vapour; but that such can ever be mistaken for pneumonia passes comprehension. True, the earliest stage of pneumonia itself is one of hyperæmia, and, practically, acute pulmonary congestion means nothing less. Forming thus a part of the disease, it can hardly be said to simulate it, since the signs that denote the presence of the state of congestion must be interpreted as indicating the occurrence of inflammation. It may, indeed, be admitted—especially in the case of children—that these signs, and the pyrexia that accompanies them, are far more transitory than we are accustomed to regard the evolution of pneumonia, and they may, in some instances, never advance so far as to denote consolidation. But we have evidence that pneumonia itself may in certain cases run an abbreviated course; that the seat and amount of pulmonary lesion may be very limited and so situated as to reveal no other signs than those of vascular engorgement and exudation (p. 237). In the adult, however, even this explanation is unneeded, since a primary acute congestion is, as we have said, practically unknown. It is otherwise with secondary or so-called 'collateral' hyperæmia, which in the presence of pneumonic consolidation of one lung may attack its fellow. Pathologically, this state is merged in that of pulmonary œdema, which is invariably a secondary phenomenon, owning many antecedents.

Œdema may arise with comparative suddenness, as when it complicates acute bronchitis, renal disease, or valvular disease of the heart. It is then signalised by greatly increased dyspnœa, cyanosis, abundant moist râles over the whole of both lungs, with perhaps dulness at the bases and enfeebled respiration, but without fever. There is no likelihood of its being mistaken clinically for pneumonia. Post-mortem the œdematous organ contrasts markedly with the hepatised. Although increased in bulk and weight, its cut surface has a smooth, glistening character, and slight pressure will cause abundant thin, serous or sero-sanguinolent, fluid to escape, in some parts frothy from admixture with air, in others quite free from such admixture. It has been clearly shown that the supervention of œdema depends far less upon conditions similar to those productive of œdema elsewhere, namely, obstructed venous circulation, than it does on a disproportion in the vigour of the two ventricles of the heart. Its occurrence indicates a weakening of the left ventricle, which can no longer keep pace with the force of the right, and is therefore unable to relieve the lungs in proportion as these become supplied with fresh blood. Hence it is, no doubt, that a certain amount of œdema is invariably to be found in the lungs after death, especially at the close of exhausting illness. This œdema is in the dependent parts of the lung, and is commonly spoken of as *hypostatic congestion* or *hypostatic pneumonia*, according to the degree of consolidation that it presents.

In the production of hypostatic consolidation it is highly probable (as pointed out by Dr. Wilson Fox, 'Atlas of Pathological Anatomy of the Lungs,' p. 41) that collapse plays a part. For we have not only to deal with cardiac enfeeblement and the tendency of the sluggish blood stream to stagnate under the influence of gravity, there is also, in most cases, a similar weakening of all the muscles of the body, and the act of inspiration is less and less efficiently performed. The condition is further aided, in certain cases, by the altered quality of the blood, favouring its exudation.

Although the physical characters of hypostatic congestion are usually distinctive enough in engorged, dark-red, semi-collapsed tissue, from which blood-stained fluid may be expressed ; yet not seldom, particularly in the aged and those who have been long bedridden, the affected part assumes more and more the characters of pneumonic hepatisation, being more firm, but yet friable, even greyish in tint, and perhaps slightly granular. It is needless to point out that we are dealing here with a 'bastard pneumonia'—a merely mechanical result of the conditions under which for some time the pulmonary circulation has been carried on. Nay, even the presence of lymph on the pleural surface does not justify us in including such cases under the head of pneumonia properly so called, for the condition is clearly a secondary one. Moreover, in most cases it passes without detection during life. What characters of inflammation it may possess, and it is not denied that it does sometimes exhibit them (see p. 207), it must owe to the state of the blood and circulation, and it would tend to confuse our conception of pneumonia were we freely to admit these *simulacra* into its category.

Pleurisy and hæmorrhage are rare with this condition of lung, while the gradual shading off of œdematous, congested tissue into that which is carnified or solid as the parts are reached which lie most remote from the influence of the flagging circulation, is strongly suggestive of a single cause of disturbance reigning through the whole pulmonary system, and more or less operative in different places in obedience to the laws of physics.

Effusion into one or both pleural sacs may concur with this form of consolidation, or take its place. In the latter case the lung will be collapsed and leathery, and often contains solid spots here and there in its substance. The section of this solid portion is smooth instead of granular, and even in the most lengthened cases grey hepatisation is exceptional.

The following is a short summary of 39 cases collected from hospital records, where long-standing illness terminated in lingering death [1] (phthisis being excluded), and exhibited in a marked degree this particular condition :—22 out of the 39 had consolidation of both lungs ; 10 of these were under thirty years of age, 9 between that and fifty, and 3 were each fifty-four. None were aged, therefore. Of these same 22, 1 only had recent pleurisy. Taking the numbers otherwise, and excluding 2 cases of erysipelas and 9 of long-standing suppuration (since in these it might be urged that the appearances were due to erysipelas or pyæmia), there remain 28 cases. Both lungs were in part solid in 19 ; the remaining 9 have one lung only solid, while the other is congested and œdematous. Again, out of these 28 selected cases, 6, at the most, showed any intimation of grey softening. Three of these were instances of fever, 2 of cancer, and 1 was without history.

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[1] It would be incorrect, we conceive, to conclude that this number represents the real frequency with which, in the circumstances stated, hypostatic congestion passes at last into consolidation. The subjects of lingering illness seek to die at home, or wanting that, they are dismissed when treatment is abandoned to the infirmary. Still, it must be admitted that consolidation is far less common with this mode of dying than the condition of œdematous congestion, which according to the hypothesis immediately leads to it. It appears further, as shown above, that this consecutive hypostatic consolidation selects rather the young and middle-aged than those advanced in years.

It would be difficult to classify these cases clinically. They were no further alike than in this, that they died, for the most part prematurely, worn out by the burden of some long-recognised disease, and that, so far as was observed, this tardy progress to the grave was not accelerated by the supervention of any acute or intercurrent affection. The list thus includes instances of slow malignant disease, of fever with exceptional lengthening out of life, of exhaustion after profuse hæmorrhage, of mechanical occlusion of the œsophagus entailing actual starvation.

Contrast for a moment acute pneumonia, as we all know it, in its striking objective signs and busy eventful history, with this chronic process of deterioration, whose insidious course is regulated by the fluctuations of a wholly distinct disease. Contrast especially the part that pneumonia plays as a local affection, invading but not destroying, and making the lung rather the field of its operations than the object of direct attack, with this gradual process of decline which awaits the period of death-agony for its full development. In whatever degree the two conditions may claim kindred on the ground of histological likeness or their common participation in an inflammatory process, yet in origin, course, and destiny, they are widely dissevered, and for all practical uses must be kept asunder.

There is yet another morbid condition usurping the name of pneumonia, whose resemblance to that disease consists mainly in this, that after a series of changes, in themselves distinctive enough, the lung becomes dense and tough, and in some instances eventually solid over a large area. The condition, which commonly affects both lungs, is best seen in the adult in cases of mitral cardiac obstruction, or indeed, of any mechanical defect producing abiding pulmonary hyperæmia.

In these circumstances there occurs a well-marked structural change, strikingly opposed in its character to what takes place as a sequel to active inflammatory congestion. Grossly, this change is indicated by the tough, leathery consistence it confers. The finger cannot penetrate the lung, and, in extreme cases, the knife grates in cutting it. Though hardly solid it is not crepitant. Its section shows a mottled surface of grey and brownish-red, with coarse white lines stretching across so as to map it out irregularly into groups of lobules. In this, its most characteristic stage, it feels like coarse muscle. The organ has lost its delicacy of texture. Essentially the same condition, as regards colour and fibrous intersecting bands, may be met with when in addition the lung is firmly solid ; or it may be that in the substance of a lobe which is tough only, a solid portion is disclosed on section. Yet always the distinction remains to separate it easily from ordinary hepatisation in its greater resistance ; it is not so solid but more coherent, breaking less but yielding more.

The histological characters of such lungs explain their gross appearances. In the first place, there is to be seen a notable thickening of the inter-alveolar tissue, as well as of the peri-bronchial and peri-vascular textures. This, which imparts the 'indurative' character to the organ, is a change precisely analogous to that observed in all other organs of the body that have been long under the influence of passive venous congestion. As Sir W. Jenner has pointed out, even the heart in such circumstances does not escape this cyanotic induration. It is not an inflammatory change, but rather a hyperplasia of tissue elements induced by the increased exudation from the blood that may be supposed to occur under conditions of long-standing venous engorgement. The effects of the obstructed pulmonary circulation are also to be seen in the remarkable dilatation and tortuosity of the pulmonary capillaries, which project into the alveolar spaces so as considerably to reduce their capacity. Lastly, the alveoli themselves are found to be partially occupied by cellular products, derived from the alveolar epithelium, and many of them filled with brownish granules, obviously the result of the diapedesis of blood corpuscles and their disintegration. Actual hæmorrhages may even be present, and free blood corpuscles found in the alveoli. It should be added that there is considerable pigmentation of the inter-lobular septa.

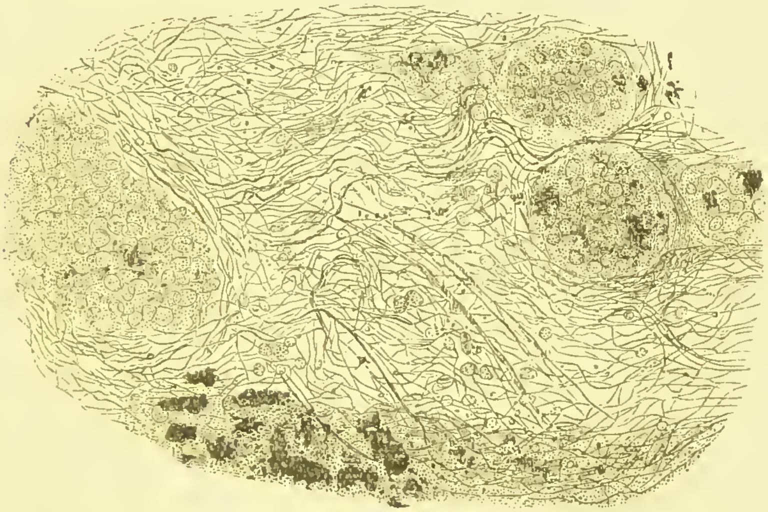

FIG. 11.—Minute appearance of tough resisting lung, from a case of mitral valvular disease, showing extensive fibroid development encircling the alveoli and separating them. The latter are seen with their contents partly granular, partly corpuscular, deeply pigmented. (×200.)

The main feature, however, of the indurated lung consists in the increase of its fibroid elements, or, rather, in the conversion of the normal elastic tissue into fibroid material (Fig. 11). By the addition thus made to the normal texture of the lung air space is encroached upon. Inter-alveolar fibroid material is multiplied at the expense of the alveolar chambers, which are thus in places almost obliterated. In an organ so far altered it needs only a slight increase or extension of the catarrhal process to render portions of it wholly airless and non-crepitant.

The agency by which this further change is accomplished is œdema. Through the intervention of a watery effusion, which itself results from impeded capillary circulation, air is displaced from the alveoli, and the lung, in those parts that œdema most affects, becomes carnified. At this period it resembles very much the compressed lung of hydrothorax, or the collapsed lobule of the child, as these appear in their later stage. And, as with them, this fluid exudation eventually gives place to variously-shaped elements, mostly catarrhal, but with a variable intermixture of exudative products. Splenisation, in the words of Rindfleisch, is 'a favourable soil for the development of such changes.' The lung is thus brought by a succession of steps, each arising naturally out of the other, to a condition of solidity more or less complete, which gets described as hepatisation and even attributed to acute inflammation.

Yet no argument is needed to separate such phenomena from those of pneumonia. In the case we are considering it is the structure of the lung that is primarily affected ; it has undergone a chronic irreparable change ; the final filling up of the alveoli is a consecutive result which—though far from invariable—the altered conditions of circulation directly favour. In pneumonia, on the contrary, normal alveoli are suddenly invaded by a plastic exudation, and the solidity which the lung acquires is due, not to itself, but to this added material.

The distinctive character of this condition appears in an analysis we have made of twenty-five cases exhibiting, post-mortem, extensive consolidation of one or both lungs, and described as 'chronic pneumonia.' Sixteen of the patients were under thirty, the oldest was sixty-two; and there was a distinct rheumatic history in ten. In at least sixteen (about two-thirds) both lungs participated pretty equally in the change which concerned most the lower lobes, often in accurately corresponding parts. Where the solidity was unilateral the fellow lung was congested, or contained patches of 'pulmonary apoplexy.' Pleurisy was exceptional and grey hepatisation conspicuous by its absence.

Now, in seventeen of these examples there was obstruction of a very definite kind, namely, narrowing (in most instances in an extreme degree) of the mitral orifice.[1] In the rest, though there was no direct obstruction in the sense of a material bar to the blood's passage, yet the circumstances warrant the assumption that a like effect was produced by the defective action of an embarrassed heart. In many of the patients a marked rheumatic history, together with youth, may fairly be presumed to have influenced the ultimate result; blood prone to deposit fibrin is placed in circumstances favourable to such an occurrence. Yet so manifest is the influence of a mechanical agency, that it seems sometimes as though an accident determined between exudation and rupture. The lower lobe of one lung will be solid, whilst its fellow is extremely congested, and contains in its midst a mass of extravasated blood. Often, as might be supposed, there is general œdema of the limbs.

Some of these cases, in their clinical incidents, strikingly illustrate both the near dependence of the condition upon mechanical causes and also its essential chronicity or state of standstill. Comparing them one with the other, the structure of the lung shows the same appearance after a week as after a month and a half.

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[1] Four or five of these cases of mitral stenosis, it must be admitted, it was not possible upon any anatomical distinction to separate from pneumonia. These are alluded to under the title of 'Cardiac Pneumonia' in Chap. X, p. 207.

Take the following examples: A young woman had had cough and dyspnœa for six months, and later, dropsy. She was admitted with that and blood-spitting, and died eventually, with no acute symptoms, after forty-six days. The whole of the lower lobe of the right lung was solid, and contained small patches of extravasation. The left lung was similarly, though less, affected. The mitral orifice was narrowed so as only to admit the little finger. The heart was covered with old false membrane.

A girl of seventeen, with obvious signs of heart disease following rheumatism, emaciates, becomes œdematous and slowly dies. Both lungs were found to be hepatised, and the heart, much hypertrophied and adherent to the pericardium, is in an advanced stage of fatty degeneration.

While these cases thus exhibit a strong family likeness, it is not to be supposed that the effect upon the lung of the lesions in question is always as uniform as it appears in these selected instances. A number of causes will be in operation besides this obvious one of mechanical obstruction, and sometimes the post-mortem appearances, as we have shown (Chap. X, p. 207), cannot be separated from true pneumonia.

The various conditions of *pulmonary collapse* are sharply distinguished from those of pneumonic consolidation. Of these we may discriminate the lobular and the lobar forms. Lobular collapse—which follows especially upon obstruction to the entrance of air, as in capillary bronchitis, croup, or diphtheria—is characterised by the depression of its surface below the level of the circumjacent lung, and the dull-red tint and fleshy consistence of the airless patch, which is tough and resistant, but quite smooth on section. Microscopically, the approximated alveolar walls, which have, in consequence, the appearance of thickening, and the scantiness of any alveolar catarrh or exudation are quite apparent. Such collapse is especially liable to occur along the thin margins of the lung, but in well-marked cases it may be distributed more or less over the surfaces (especially the posterior). The absence of air leads to an apparent increase in the amount of blood in the collapsed region, which may perhaps pass into an actual turgescence, and account partly for the subsequent development of alveolar catarrh and pneumonic foci, so often described. But that collapse is the invariable antecedent of catarrhal or broncho-pneumonic consolidation may well be doubted. An essential distinction between collapse and catarrh is the readiness with which the former areas may be reinflated with air.

Of collapse on a larger (lobar) scale, our examples must be sought mainly in compression from pleuritic effusions and pneumothorax. There can be no possibility of mistaking the toughened, pigmented, flaccid lobe, with its shrivelled, wrinkled, and thickened pleura, for a hepatised lung. The fleshy 'carnified' tissue has no counterpart. Whether it has been so recently produced as to be capable of reinflation, or so long compressed and altered as not to yield this evidence of its nature, there is seldom need for making the test. Clinically, a lung compressed by pleural effusion may present physical signs indistinguishable from consolidation, tubular breathing included (p. 54, note); but it is rare for these signs to be so associated as to cause any hesitation in determining the diagnosis. Conversely, a pneumonic lung, of which the bronchi are plugged by exudation, may, as we have seen, yield the negative signs of pleural effusion (p. 55, note). But in either case the circumstances are exceptional, and other factors come into consideration.

PART III

ETIOLOGY AND PATHOLOGY

CHAPTER XV

ETIOLOGY.—I. WEATHER AND CLIMATE

Many 'extrinsic' causes of pneumonia are predisposing—Comparison with bronchitis—Seasonal (monthly) incidence—Cold *per se* not sufficient—Rainfall—Wind—Discrepancies in statements on this head—Barometrical pressure—Imperfect knowledge of relationship between meteorological conditions and the disease.

In pathology, as in every department of science, the progress of research has led to the disturbance of many a deep-rooted dogma. Especially is this the case in that branch of pathology which deals with the causation of disease. From one point of view, indeed, advancing knowledge upon this subject seems to render it more complex than ever, raising many new problems for solution; whilst, on the other hand, a way is opening to the simplest explanation of the genesis of many diseases by reference to the operation of external microscopic germs.

If we separate intrinsic from extrinsic causes, we have to determine, in the first class, what has produced the state of the organism which favoured the development of the disease, and, in the second, to ascertain how outside influences operate upon the body. In neither case have we, as yet, reached anything like exact knowledge. We have but advanced so far as to replace the intrinsic agencies by the extrinsic, and thereby, in a sense, to revive the notion of disease being introduced into the body from without. Not, as was thought in old time, that disease is an entity, which, having once gained entrance, needs to be expelled by force, but rather that the disordered state of the economy, brought about by the operation of external agents, is directly responsible for exciting particular lesions. Even then, we are bound to admit the intervention of conditions—so-called predisposing causes—which render the organism vulnerable.

In pneumonia we have a striking instance of this change in respect to our etiological conceptions which modern inquiry seems to demand. Exposure to cold—chilling of the surface—was, until recently, universally regarded (and still is by many) as the sole efficient agency for producing this disease, which was accordingly looked upon as a local inflammation. No one can deny the very close relation that such a condition often bears to the invasion of pneumonia. Nevertheless, the doctrine in its bald simplicity is no longer tenable, even when the view is limited strictly to cases where the operation of cold seems to be most marked. Take another doctrine (or dogma), namely, that pneumonia attacks, even preferentially, the strong and robust. We may not be able to meet this with a universal negative, but there is evidence in plenty to show that in a vast number of instances the subject attacked by pneumonia is, at the time, debilitated in some way or other; or, in other words, is rendered more susceptible to the influences which produce the disease. The preference shown by the affection for the very young and the aged, or those who are the victims of alcoholic excess, or subjected to insanitary surroundings, or who spend their lives in sedentary pursuits, is a matter of fact; and unless we are prepared to multiply without limit the varieties of the disease, according to each separate circumstance of its origin, we are bound to admit that 'predisposition' is a most potent factor in its production. On this ground, then—to revert to the question of chill—we may surely regard the influence of exposure to cold as one out of many secondary or predisposing conditions under which this disease arises. In what manner chill operates is practically undetermined, but no one can doubt that exposure to extremes of temperature does lower the vital resistance of the organism. The further consideration of this subject will, however, come more fitly after we have reviewed in detail the relationship of pneumonia to meteorological and climatic influences. Other etiological factors will then need consideration, including the evidence that has been brought forward of late years in support of the specific nature of the disease, of which, upon that view, the pulmonary inflammation is but a local manifestation.

The general influence of weather upon the so-called inflammatory diseases of the chest is matter of common observation. Low temperature, easterly winds, sudden changes of whatever kind, are believed to increase the whole group of chest affections alike. We propose to consider how far this popular view is strictly correct; for it seems not unreasonable to expect that diseases so different in their history and progress as pneumonia and bronchitis should in their origin be influenced in different degrees by the same set of circumstances.

In approaching this question it is above all things necessary to divest the mind of preconceptions; the point should be capable of statistical demonstration. Thus, one of the highest authorities (Jürgensen), who fully recognises the marked influence of the season of the year in the prevalence of pneumonia, does not hesitate to declare that it is influenced very differently from pleuritis and bronchitis. Nevertheless, the figures quoted by him from the London mortality returns and the records of the Vienna Hospital for twenty years hardly bear the construction he imposes on them. Even the diagram he gives (see Fig. 12), based on the latter statistics, shows fairly enough a correspondence in the rise and fall of catarrhs and pneumonia during the several seasons; a correspondence which is most strikingly proved to exist by other writers, notably Seibert and Baker. It would almost seem as if the argument that chill can play no part in producing pneumonia had been allowed to override the general fact of the coincidence between the prevalence of that affection and of diseases more directly referable to exposure, whereas, the case for a specific agency at the root of pneumonia, which is the contention of Professor Jürgensen, is not strengthened by discarding chill as one factor in its etiology.

Any meteorological inquiry to be complete must review the weather conditions, both in this country and abroad, at several periods; at times when the diseases in question are unusually prevalent, and at times when they are unusually rare. Even were the materials at hand for so wide an investigation, it would obviously be open to many sources of error. We have, for instance, to trust to reports of very unequal value. For our own country there exists no fuller information than that furnished by the Registrar-General's Reports, and of the Reports of other countries it is difficult to estimate the comparative value. And while we desire to deal only with the primary forms of the diseases concerned, there are no means, in such figures as we get, of separating these from the others. Further, it does not follow that the prevalence of a disease is always correctly measured by the number of deaths it occasions. There is, besides, the difficulty of estimating the period which should intervene between the weather, which is the assumed cause, and the death, which is the ultimate result. The mortality from pneumonia or from bronchitis suddenly rises; at what precise date are we to look for the circumstances of weather to which that increase is due? In our ever-varying climate it must always be hard to determine this point, or even to say whether it is the weather of some particular period, or the mere fact of change of weather, which produces the result. These and numerous other sources of fallacy tend to disappear in a multiplication of instances. It is at least worth trying whether (regarding the Registrar's tables as true *comparatively*, and not at all for absolute numbers) it may be possible by a series of observations upon the meteorological phenomena which attend periods of high and of low mortality from pneumonia, to arrive at some general conclusions as to the influence of weather; conclusions which must either stand or fall according to what we learn of the distribution of the disease throughout the globe, of the circumstances attending its epidemics, and of such histories of individual cases as our own experience may furnish.

Accepting, then, thus far, the information of the Registrar-General's Weekly Returns, it will be observed, in the first place, that under the *influence of weather* (and especially of cold) the whole number of acute chest affections are at least *similarly* affected; their death rates rise and fall together. The only question is whether, so far as this evidence goes, any particular condition of weather affects them unequally; so that, for instance, we may say that cold is especially prejudicial to one disease, easterly winds to another, and so on. In the case of pneumonia, the fact, which soon appears, that there is a near correspondence between its fluctuations and those of bronchit adds, no doubt, to the difficulty of determining the precise conditions whose influence is unequal in the two cases. At the same time, if these two affections vary as to their death rate simultaneously, we may safely conclude that the causes on which these variations depend do the same; that they are to be looked for at one and the same time for both diseases. The objection, therefore, that to compare the death rate of bronchitis with that of pneumonia is to compare diseases differing in duration, which, if they end together must have started far apart, loses its force. In every week, no doubt, deaths occur under these two headings after very different periods of illness, and from causes quite remote from climate · yet, on the whole, for purposes of comparison, we may assume from this strict harmony between the two that the fluctuations in the rates from week to week express truly the effects of the same period of weather in both cases.

Now, it appears that the lowest mortality of both pneumonia and bronchitis falls in the latter part of summer, and the highest of both during the winter. The proportion of deaths, however, between the two diseases is not constant throughout the year. During the five or six temperate months, say from May to October, this proportion approaches nearest to 1, it being not unusual at midsummer for the mortality from pneumonia even to exceed that from bronchitis. But about the latter end of October, or thereabouts, sooner or later, according to temperature, the bronchitis rate rises in a greater degree than the pneumonia rate, and the maximum of difference for the two is obtained about January or February. It will be found, moreover, that while the minimum of deaths for both occurs at or about the same time, namely in August and September, the maximum of deaths is earlier in the case of pneumonia than in bronchitis. Thus, taking the average of ten years, 1857–66, the highest number for bronchitis occurs in the middle of January, while for pneumonia it is nearly two months earlier, that is at the end of November.[1] We have, therefore, these two diseases presented to us as of unequal range, that of bronchitis being the wider. In summer the causes provocative of both are at their weakest; as winter approaches these are more felt, and pneumonia is apt to suffer to its utmost earlier in the year than bronchitis.

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[1] In Paris, according to Grisolle's observations, September yields the smallest and April the largest number of cases of pneumonia In Vienna

Such are the conclusions drawn from our own Registrar-General's Report. Jürgensen [1] finds that the *monthly distribution* of pneumonia is slightly different in a continental and in an insular climate. Thus, in the former, he says that the maximum frequency of the disease is from March to May, and the minimum from September to November; whereas in the latter, the maximum falls from December to February, and the minimum from July to August. Seibert,[2] as a result of a collective investigation among fifty observers in New York, carried on from March 1, 1884, to March 1, 1885, found that for that year, February and March yielded by far the largest number of cases, viz. 243 out of a total of 768; and July and August the smallest, viz. 51 cases. Many other statistics have been published, some referring to single years, others to periods of many years. Thus Kohnhorn [3] gives among 321 cases observed during nine years—1873–1881—a great increase in March, April, and May, with a rapid fall to the minimum in July to September. Port [4] and Keller [5] with 370 and 363 cases respectively, extending over nearly the same period, arrive

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the disease was most prevalent in the first five months of the year, especially in March, April, and May. See 'Reports of Vienna Hospital,' vol. i. 23, 1860, 61, 62, Schmidt's 'Jahrb.' Taking the numbers for Paris and London with a view to compare the rates from bronchitis and pneumonia respectively, we find often a striking difference in the proportions: thus—London, week ending March 12, 1870, bronchitis, 305; pneumonia, 100. Paris, week ending March 5, 1870, bronchitis, 100; pneumonia, 131.

[1] Ziemssen's 'Cyclopædia' (Amer. ed.), v. p. 12.

[2] 'Berliner klinische Wochenschrift,' 1886, p. 269.

[3] 'Vierteljahrschr. f. gerichtl. Med.' July 1881, quoted by Seibert.

[4] 'Archiv f. Hygiene,' 1883, quoted by Seibert.

[5] In 'Croupöse Pneumonie,' Tübingen, 1883, edited by Jürgensen.

at very nearly the same result; as the curve constructed by Seibert from their combined observations shows. This curve corresponds fairly well with that given by Jürgensen (Fig. 12), based on twenty years' statistics of the General Hospital, Vienna, and that by Baker, showing the prevalence during eight years (1877–84) in Michigan (Fig. 13), and also with one by Edlefsen,[1] giving the results of thirty-four years at Kiel.

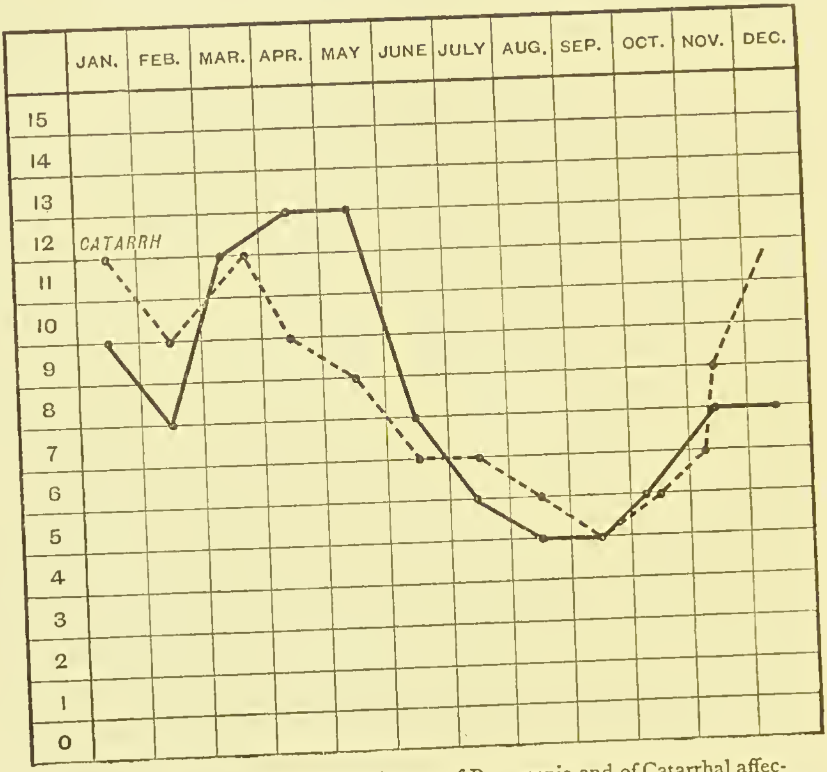

FIG. 12.—Monthly variation in incidence of Pneumonia and of Catarrhal affections, based on 11,913 cases treated at the Vienna General Hospital during twenty years (after Jürgensen).

It will be seen that, although there is undoubtedly a general correspondence between the rates of prevalence of bronchitis and pneumonia, the parallelism is by no means exact, and their divergence is more strikingly seen when the matter is viewed, not merely from the side of seasonal variation, but with reference to the prevailing meteorological conditions. For although—as clearly demonstrated by Seibert and others—the occurrence of pneumonia is markedly influenced by weather, it does not appear to hold precisely the same relationship to weather-conditions as catarrhal affections do. The difference, it must be admitted, is not striking, and the author disregards it, considering, indeed, that catarrh predisposes to pneumonia.

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[1] In debate on 'Die genuine Pneumonie,' Congress für innere Medicin. Berlin, April, 1884.

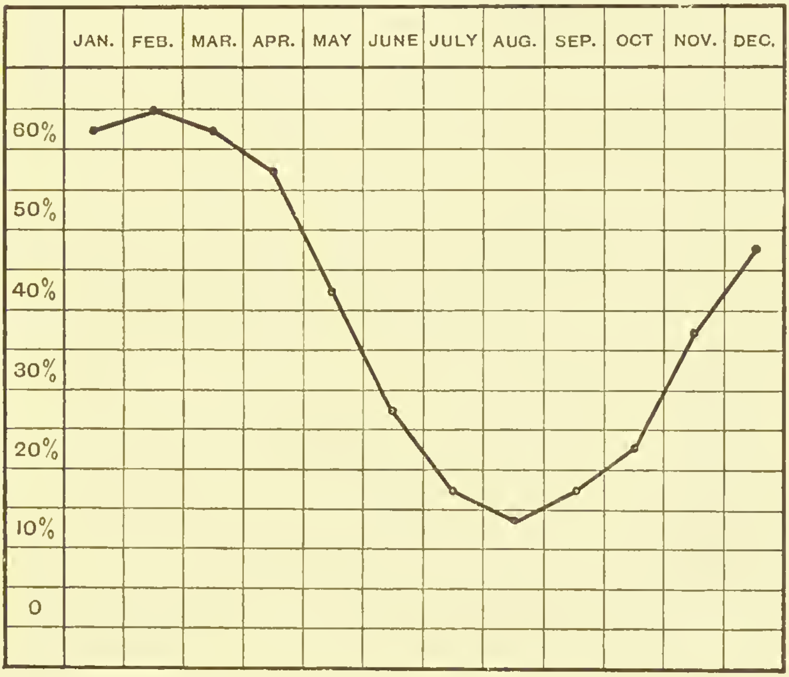

FIG. 13.—Monthly variation in incidence of Pneumonia, from returns made in Michigan, 814 cases (from Baker).

As regards the influence of *temperature*, it would seem—to revert to our own returns—that cold does not necessarily affect the pneumonia rate, but always and markedly the bronchitis rate. Take for instance the early winter of 1859 : a low temperature towards the end of October and again in the middle of November is followed by a bronchitis rate greatly in excess of the average. Pneumonia, on the other hand, though not unaffected by the change, is only slightly so, it remains to the end of the latter month much under its average, when, with excessive cold and a north wind, it rises considerably. Again, extreme cold in the middle of January 1867 is followed by a large increase in the rate of bronchitis, which, for the week ending on the 26th, rises to 162 over its average; while pneumonia actually decreases at the same time, and, to the end of the month, remains below its ten years' average; the direction of wind being north-west and south-west.

A glance at the tables will show how surely the rate of bronchitis is affected by cold. They show, no doubt, a similar tendency on the part of pneumonia; but, as has been shown, this is not invariable nor strongly marked. There seems, indeed, no assignable limit to the rise of bronchitis with lowered temperature. With the extreme cold of January 1864 its death rate (for the week ending January 16) is perhaps the highest on record; pneumonia, on the other hand, many times during the same year is higher than for this particular week.[1] It has been said already that pneumonia attains its maximum mortality in November. If cold were the chief agent in its production it would, like bronchitis, be most frequent in January.

This conclusion as to the effect of cold receives support from what we know of the geographical distribution of pneumonia. It is a disease especially of temperate regions. At the poles and in the tropics it is very rare. It has been computed that the proportion of pneumonia to other diseases at Chamounix is as much as 1 in 5, while on the African coast it is as little as 1 in 627. It is very rare in the Presidency of Madras; equally so amongst those exposed to the extremest cold in expeditions to the Arctic regions.[1]

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[1] It may be urged with truth, that the general effect of extreme cold in carrying off old people is here to be taken into account, since these, although dying with some form or other of bronchorrhœa, are not in fact killed by bronchitis, although, for want of a better word, that cause of death is assigned. But even omitting these, or a large proportion of them, the above remarks would still hold.

Does *rain* influence the rate of mortality from bronchitis or pneumonia? We have no sufficient data upon the subject, but such as we have yield a very uniform result. Take, for example, the year 1859. In the weeks following periods of abundant rain it happens always that the disproportion in the mortalities of the two diseases is conspicuous. Thus, in the week ending July 2, 1·24 inch of rain fell; for the following week bronchitis is above and pneumonia below its average. In the week ending July 23 there were 2·15 inches of rain. For the subsequent week the mortality from bronchitis is the highest, and that from pneumonia is the lowest of the whole ten years of corresponding weeks quoted. There is, in fact—it so happens—only one week in this year where a great fall of rain is not followed by a similar altered proportion, and on this occasion nearly all the rain fell on one day, a steady downpour of nine hours. Take 1867. Here again it will be found that after weeks of unusual rain bronchitis increases out of proportion with pneumonia. Other years might be quoted to the same effect. There are, of course, numerous circumstances to be considered before asserting that wet weather has any direct influence of this kind; but probably no more than the truth is expressed by saying that any considerable amount of wet has a tendency to heighten the bronchitis rate, but has no such tendency as regards the pneumonia rate; the very lowest numbers of this latter out of ten years being found to follow weeks of excessive rain ('Weekly Returns,' vol. xxviii. pp. 198 and 321).

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[1] 'British and Foreign Med.-Chir. Review,' xxii.

As bearing upon this question, we find that in marshy localities, where intermittent fever is prevalent, pneumonia is little felt, although bronchial catarrh is often common in such places. We know, too, that during the rains of the monsoon in India pneumonia shows a remarkable decrease. According to an analysis by Dr. Morehead of 313 cases admitted into hospital, it appears that the three months of greatest rain—July, August, September—are the three also during which there is a remarkable decline in admissions from this cause. The numbers are the smallest of the whole year, and July, the very rainiest month, shows only nine such admissions, the next smallest number falling in August, and being just double of the month before.[1]

In judging of the apparent influence of *wind*, it is, of course, not enough to regard only the quarter of the compass recorded by the register; we must consider the force of the wind as well as its direction. At all seasons of the year there are many days when, from the air being comparatively at rest, we are unconscious of the fact that the wind, such as it is, is setting from the east or north-east; there is no wind properly so called. The proverbial belief of mankind which connects various ills to the body with the blowing of an east wind takes its origin from our own sensations when exposed to such blasts. It is not supposed—probably it is not true—that the same or similar effects follow when these sensations are not experienced. Calm weather or a small amount of horizontal movement of air is apt to correspond with low death rates from pneumonia, be the position of the weathercock what it may. Wind, on the other hand, it would seem, is favourable to the production of pneumonia, chiefly, if not altogether, when its direction is northerly or easterly. It is only an illustration of this remark to find that, during the calm weather of the spring of 1859, although the direction of the wind was registered as north-east, the mortality from pneumonia was unusually low. With the wind in that same quarter and a large amount of horizontal movement (as in March of 1867 and August of 1866) the mortality greatly exceeds the average. If we say, then, that high winds from the north and north-east, combined with a low temperature, yet not necessarily very low, favour the development of pneumonia, and that bronchitis, while similarly affected by the same causes, suffers in a less degree, the statement will be borne out by the returns, while it may be added, that the effects of a very low temperature and of much rain are seen in raising the bronchitis rate rather than the pneumonia rate.

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[1] Dr. Morehead, comparing his figures with those of M. Grisolle, observes that for these months there is a smaller decline for Bombay than for Paris; he argues thence that a moist atmosphere and high winds tend to produce pneumonia. The inference is surely unjust. Dr. Morehead's tables render it probable that the range of pneumonia in Paris is greater than its range in Bombay. They show at the same time that the causes productive of the disease in Bombay are at their least during these monsoon months. If, then, rain is the great feature which distinguishes this season from others, we are forbidden to attribute to it any special influence for evil. Dr. Morehead on the 'Diseases of India,' vol. ii. p. 308 *et seq.*

It may be said that observations on several agencies taken separately are open to objection. Let us consider them in combination. We may choose, for instance, a year of very high mortality so far as these diseases are concerned, and compare it with one of very low mortality, and so, by placing the weather phenomena of the two years side by side, discover the conditions on whose presence or absence the variations in the death rate apparently depend. Now, it happens that the summer of 1859 was remarkable for a very low mortality from pneumonia, and the summer of 1866 for a very high mortality from the same cause. In the former year the bronchitis rate is not affected; in the latter it is high, but not raised in an equal proportion with pneumonia. As regards the state of the general health, the total of deaths for both years exceeds the average, owing to a large mortality from diarrhœa. In 1866 there was a short outbreak of cholera, which, commencing in July, was at its height at the opening of August, when 1,053 deaths are registered for one week. From that time it declines considerably, but still numbers between 100 and 200 died weekly until late in the autumn. At the worst of the epidemic we have pneumonia showing a higher mortality than bronchitis, both being above the average. A similar phenomenon is observable in other cholera years, and especially in 1849.[1] On the other hand, in the year 1859, with a severe epidemic of diarrhœa, but with very little cholera, there is an almost unprecedented fall in pneumonia, in which bronchitis does not share.[2]

What are the meteorological circumstances concurring with the low and the high rate specially affecting pneumonia in these two years respectively? Both summers are exceptionally hot. With the low rate of 1859 the wind is chiefly southerly, less variable, and for a fewer number of days easterly and north-easterly than with the high rate of 1866. The horizontal movement of air throughout the months referred to, July and August, is much less for the healthy year. Furthermore, with the year of high mortality, the wind veers round to the south-west in the week ending September 8, and the pneumonia rate becomes less for the week following. In fact, the great disparity between the rates of the two years disappears for a while at this point, and so long as, for both years, south-west winds are blowing. In October, however, with a return to the conditions of the summer, that is to say, variable *east* winds for 1866 against less variable *south* winds for 1859, there is again a wide difference. The healthier autumn is also much the colder; south-west winds and a small horizontal movement still coincide with the low rate.

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[1] See, for instance, weeks ending August 4 and August 11 of 1849.

[2] See especially weeks ending July 30 and August 13, 1859.

Again, place the week ending November 12, 1859, beside the week ending on the same day of 1864. We choose these two only because they exhibit the largest difference we can find; for 1859 the deaths from pneumonia are 60, for 1864 they are 165. Bronchitis, again, does not sympathise in the case of the low rate, but it does in the case of the high. There is no severe epidemic in either year, but typhus is twice as fatal in 1864 as in 1859. Taking the weather of the latter part of October in each of these years, it appears once more that the healthier year for pneumonia is by far the colder—the temperature, indeed, of the week ending October 29 is only 38·6; it is unprecedentedly low—far lower, at least, than any other year shown. The effect of this low temperature is not unmarked; pneumonia rises considerably, yet for one week only, and then not so high as to reach its average; bronchitis, on the contrary, rises from much below to much above its average, and continues to rise during subsequent weeks. From the middle of October to November 5, 1859, the period of the low rate, the wind is chiefly south-west. That period, therefore, contrasts remarkably with the high rate of the corresponding time in 1864, when the direction of the wind is north and north-east. There is more rain with the low pneumonia rate than with the high.

Once more, we find included in one and the same year—the year 1868—a remarkable contrast in comparing similar seasons, February showing a very low rate from pneumonia, and December a very high rate. Thus the deaths from pneumonia for the week ending February 15 are 58, and from then till the end of March the weekly numbers are just over 70. In December, on the contrary, the rates for three successive weeks are together 360, as against 207 for three weeks of

February. Now, the temperature during the last weeks of January and of November (the periods respectively preceding the low and the high rates) is about the same, but the beginning of February is colder than the beginning of December; still, therefore, we have the greater cold with the healthier time. The direction of the wind from January 26 to February 15, that is with the low rate, is mostly south-west and west-south-west. It is the same during March; and during March there is again a low mortality in pneumonia.

If the weather for this same year be followed throughout a similar rule seems to prevail. Thus for the first half of April the wind is easterly; by the 18th of that month the mortality has risen to 98. As regards the high rate which distinguishes the latter part of the same year, we have the wind variable from November 22 till the end of the month, then east and south-east till December 3, when, after a few days of south-westerly winds, it again becomes variable, but chiefly south-east till the 15th. At this date it sets south-west, and continues in that quarter with hardly a break till the end of the year. And now the supposition that this heightened mortality of early December is somehow connected with the direction of the wind is rendered probable by the fact, that with the change to south-west we have for the last two weeks of December—colder weeks and very rainy—a mortality of only 170 against 243 for the first two weeks.

It is satisfactory to find in corroboration of these statements that the Returns of the Registrar-General for Ireland are very similar. Drs. Grimshaw and Moore, who with much pains have investigated this subject,[1] find that for the four quarters of the year the mortality from bronchitis is markedly influenced by temperature; it was twice as great in the first as in the second quarter, and more than four times as great in the first as in the third quarter. The mortality from pneumonia, however, was only one-fifth greater in the first than in the third quarter. The extreme winter fatality of bronchitis and its low summer fatality are equally wanting in the case of pneumonia.

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[1] 'Dublin Journal of Medical Science,' March 1875.

The further statistics of these authors in reference to Dublin, as regards the influence of rainfall and of dry north-easterly winds, are still in strict accord with those we have quoted.

Since these results, gathered from the returns of the Registrar-General, were published in the first edition of this work, the subject of the influence of weather in determining pneumonia has been studied by many other observers, with the advantage of dealing with the actual prevalence of the disease and not with its mortality alone. It is incumbent upon us to refer to their conclusions, and also to those which arose out of the inquiry by the Collective Investigation Committee upon this point, especially as there is not perfect unanimity among these authorities.

Dr. A. Seibert, of New York, from his study of the subject,[1] has arrived at very definite conclusions regarding it. He believes that the weather conditions favouring catarrh of the respiratory mucous membrane are also those which most influence the production of pneumonia. According to him it is the sudden occurrence and long persistence of damp and cold that are especially harmful. He demurs to the conclusions arrived at by others, by showing that they are based either on imperfect observation, or insufficient appreciation of all the bearings of variable meteorological states; and points out that the question can only be thoroughly and scientifically decided by the collation of a large amount of cases within a comparatively short space of time, where the meteorological conditions have been exactly noted from day to day, and in relation to one another. In his third paper he gives the details of such an inquiry, based upon returns of 768 cases made by 50 observers in New York, from March 1, 1884, to March 1, 1885, and we have already alluded to the results of this inquiry in reference to the monthly prevalence of the disease. He points out that whereas in winter, spring, and autumn there was a distinct parallelism between pneumonia prevalence and rain (and snow) fall, in the summer the disease remained at a low level, although during July and August the rainfall was great, and in September very small. But he subjects the weather-conditions of the whole year to a closer scrutiny, and shows that even diurnal variations are followed by a rise or fall in the occurrence of pneumonia according to the meteorological state then prevailing. The favouring conditions are 'low and falling temperature, high and rising humidity, and high wind,' each of which *per se* seems to favour pneumonia, but when two of them are associated (*e.g.* high humidity and low temperature, or falling temperature with high wind) the prevalence is greater, whilst, if the three factors are present at the same time, pneumonia is exceptionally frequent, and continues to prevail so long as those states of weather persist. Since precisely the same conditions are associated with undue prevalence of catarrh, Dr. Seibert would regard catarrh as the immediate predisponent to pneumonia—the connecting link, in fact, between the chill and the specific disease. Thus does he attempt to harmonise the old doctrine with the new.

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[1] 'Amer. Journ. Med. Sci.' January 1882. 'Berl. klin. Wochensch.' 1884, p. 273. *Ib.* 1886, p. 269.

It will be seen that, in some respects, these conclusions differ from those arrived at by others who yet fully admit that weather conditions have considerable influence in the causation of pneumonia. The latest contribution to the subject is a paper[1] by Dr. Henry B. Baker, of Lansing, Michigan, based on an elaborate statistical analysis of the prevalence and mortality from pneumonia, in Michigan, ranging over several years, and compared with meteorological observations during the same period. Dr. Baker points out that it is dryness rather than humidity of the atmosphere, which, associated with cold, or with high wind, favours the disease; for he remarks that, although, as a rule, relative humidity is high in cold seasons, the *absolute* humidity is lower then than in hot weather. His statistics show that the prevalence of pneumonia has not much relationship to relative humidity; whereas, when the absolute humidity is high (as with the temperature) the cases of pneumonia are few. Upon this, Dr. Baker bases an ingenious theory of the production of the pulmonary inflammation by the excessive exhalation of aqueous vapour over that inhaled, and the deposit of salts, chiefly chlorides, in the lung, where it acts as an irritant. His argument need not be pursued, for it is obvious that, although it may seem to have some support in the fact first noted by Beale, that the excretion of chloride of sodium by the kidneys is much reduced or even held in abeyance during a pneumonic attack, and that the exudation in the lung is charged with this salt, the theory does not explain the limitation of the inflammatory lesion to portions of one or other lung.

E. Masson,[2] of Berne, endeavoured to ascertain the precise meteorological conditions favouring pneumonia at Berne and Neuchâtel. In reviewing the authorities upon the subject, he shows how widely they differ. Thus, Huss and Hirsch believed that a locality exposed to wind was more liable to be the seat of pneumonia than one not so exposed; whilst

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[1] 'The Causation of Pneumonia,' reprint from the 'Annual Report' of the Michigan State Board of Health. Lansing, 1888.

[2] 'De l'Influence des Conditions météorologiques sur la Production de a Pneumonie,' Inaug. Dissert. Berne, 1879.

Ziemssen and Jürgensen do not admit this. Hirsch, Jürgensen, and Oesterlow thought cold played only a subordinate part, whilst Huss, Huller, and Lebert laid stress on its action in producing the disease. Similar discordant opinions are expressed as regards the influence of thermometric and barometric and hygrometric variations. Masson's own observations upon 400 cases deal with a large number of careful meteorological data. He found that, both at Berne and Neuchâtel, there was more pneumonia when the temperature was low, but that the state of the barometer had no appreciable influence. As regards humidity, there was a curious difference in the two places; for whereas at Berne a low range of humidity seemed to favour the development of pneumonia, at Neuchâtel the reverse was the case. At Berne the influence of the north wind was marked, but at Neuchâtel, sheltered at the foot of the Jura, it was less obvious. He concludes that the meteorological conditions favourable to pneumonia are varied and multifarious, the most constant being low temperature, low humidity, and the prevalence of north winds.

It is hardly necessary to pursue the subject further,[1] especially as upon certain points, *e.g.* what constitutes 'humidity,' there is a singular want of agreement amongst writers, and it must suffice to affirm that pneumonia is not, as some now hold, entirely independent of climatic influences; nor yet, as others affirm, solely to be referred to such conditions. Upon this point there are no material changes to be made in the conclusions adduced above from a comparison of the Registrar-General's returns with the weather statistics of this country.

In the inquiry by the Collective Investigation Committee of the British Medical Association[2] questions were put to the observers with a view to information upon this subject. As regards the locality of the patients' dwellings it appeared (*a*) 'that in 957 returns, 565, or 59 per cent., stated that the pneumonia occurred in localities seated on *high* ground, and 392, or 41 per cent., on *low* ground; that (*b*) amongst 885 returns, 486, or nearly 55 per cent., state the nature of the soil to be *dry*, and 399, or 45 per cent., to be *damp*,' whilst (*c*) 'of 753 returns there were 507 which gave the situation as *exposed*, or 67·3 per cent., and 246 to be *confined*, or 32·7 per cent.' Again, the larger proportion of cases occurred during a prevalence of east wind, but the greatest mortality during north winds. It was concluded that 'the disease is more prone to occur in exposed situations, subjected to the east or south-west winds, and in cold, wet (or damp) seasons.'

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[1] See especially Kohnhorn, *loc. cit.*, Port, *loc. cit.*, Keller, *loc. cit.*, Senfft, 'Berlin. klin. Wochenschr.' 1883, No. 38.

[2] 'The Collective Investigation Record,' vol. ii. July 1884, p. 36.

Hospital statistics, though in themselves much more reliable than these, exhibit numbers too small to be safely dealt with. Speaking generally, the prevalence of pneumonia during the summer is no uncommon experience in our London hospitals. Of one of them—St. Thomas's—Dr. Peacock says:[1] 'In the last fourteen years the largest number of admissions was during spring and summer. Taking a more limited period, 10 cases were admitted in the winter quarter, against 20 in the spring and 22 in the summer quarter: facts pointing,' as Dr. Peacock believes, 'to the production of the disease rather by sudden alternations of temperature and chills when the functions of the skin are active, than to severe, though more continuous, cold.'

The season incidences of 247 patients admitted to the

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[1] 'St. Thomas's Hospital Reports,' vol. v. p. 5. A year later (June 1875) pneumonia was more frequent in the Westminster Hospital than at any previous time of the year.

Westminster Hospital in five years (1884–1888)[1] is as follows—reckoning by the usual quarters of three months each :

First quarter (January–March) . . .	14, 8, 22, 12, 15
Second quarter (April–June) . . .	29, 7, 11, 17, 19
Third quarter (July–September) . . .	8, 7, 8, 15, 8
Fourth quarter (October–December) . .	8, 14, 11, 7, 7

giving totals for the whole period in the successive quarters of 71, 83, 46, 47.

If the Report for 1889 (not yet printed) be added, the total numbers during *six* years are 298, and the distribution for successive quarters 89, 93, 54, 62 ; that is to say, 182 patients for the first half of the year, and 116 patients for the second half. Perhaps the only safe conclusion to be drawn from these figures is that the first half of the year is more productive of pneumonia than the second ; but it may be mentioned that the third quarter would have given much the smallest number of patients were it not for one exceptional year, 1887, when the entries were about double of those for the other five years.

During the five years 1884–1888 there were admitted into the Middlesex Hospital 257 patients with pneumonia.

The numbers for the successive years in each quarter are as follows (the hospital, it should be stated, was closed during part of the third quarter of 1887, which accounts for the reductions for that quarter average) :

First quarter (January–March) . . .	16, 18, 16, 14, 24
Second quarter (April–June) . . .	14, 19, 17, 18, 19
Third quarter (July–September). . .	4, 7, 4, 1, 7
Fourth quarter (October–December) . .	15, 10, 13, 12, 9

giving totals for the whole period in the successive quarters of 88, 87, 23, 59 ; that is to say, 175 for the first half of the year, and 82 for the second. The numbers accord generally with those of the Westminster Hospital, but here the three months

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[1] See 'Westminster Hospital Reports,' vols. i.–v.

July, August, September, stand out more conspicuously as the period during which pneumonia is at its lowest.

A sum of the two series, including the sixth year of the Westminster Hospital, as above, would give a total of 555, distributed thus :

First quarter	177
Second quarter	180
Third quarter	77
Fourth quarter	121

or 357 for the first half of the year, 198 for the second.

The frequency of pneumonia at great elevations may depend upon conditions which we do not now discuss. We are told that pneumonia and pleurisy take the first rank amongst the diseases of the most elevated towns and villages of Europe. From numerous observations M. Lombard is led to believe that the prevalence of inflammatory maladies of the chest is in direct proportion to the elevation of the place above the sea-level. It is stated further, that the spring is their time of greatest frequency ; the sickness increases as the snow melts. Moreover, epidemics of pneumonia, such as have been often observed in the mountains of Switzerland, belong more to the high valleys than the low, whilst the reverse is the case with regard to epidemic catarrh.[1]

These details are tedious, and must fail to be wholly convincing. There is a difficulty in rendering them even intelligible without appending the tables to which they refer. Such as they are, the conclusions to which they point seem to be in accordance with the little that is to be learnt regarding the distribution of pneumonia throughout the globe. We know that neither extremes of temperature, nor swamp, nor moisture, nor the climates which are most unfriendly to human life, have any marked influence in producing the disease in question. It is in the regions that are called temperate and in their exposed places that pneumonia is especially prevalent. Where north and north-east winds prevail, where the configuration of the country is favourable to currents of cold air, in such places as Madrid, Genoa, Florence, Naples, Gibraltar—different in other respects, but alike in this—in the high plateaux of countries whose lowlands are strangers to the disease, as in Africa and Mexico, pneumonia finds its chief victims. Shall we be very far wrong, then, in concluding that the brisk movement of air at a moderately low temperature (which perhaps might be proximately determined in degrees) is amongst the most obvious exciting causes of simple pneumonia—cold, dry, penetrating winds from the north and north-east for pneumonia; a greater degree of cold, wet weather, variable winds, for bronchitis?[1]

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[1] Lombard's 'Climats des Montagnes,' p. 70 *et seq.*

Observations such as these are likely ever to fall far short of demonstration, and in fact they refer but to a small part of a very large subject. The etiology of pneumonia must comprehend all antecedent states of the body which render it assailable from without. And, however it may seem to us that the disease is brought about by such circumstances of weather as those we have been trying to investigate, we know that it is not wholly so; that its root lies deeper than these, that they do not of themselves call it into life.

Even so conditions of weather and season are far too complex, and the interdependence of meteorological states too little understood, to enable us to deduce any but the broadest conclusions with respect to the causation of pneumonia from this standpoint. It is apparent that the observations we have cited are not so harmonious as to enable us to define precisely the relationship between weather influences and pneumonia. It is not merely the seasonal incidence of the disease that has to be taken into account, even when the statistics of many years are collated; for, as all workers in this field of inquiry admit, numerous fallacies underlie such reckoning. What is required, as Seibert truly points out, is as close a study as possible of every variation in weather side by side with the varying prevalence of the disease, and even this would be of small account unless the observations were undertaken over wide areas and in many climes.

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[1] It is remarkable that at Gibraltar, where pneumonia prevails during the greater part of the year, the west winds of November and January concur with a greater frequency of the disease than do the east winds of other months. But then the east winds are described as bringing along with them much humidity and fog, while the west winds are cold and dry. Grisolle, 'De la Pneumonie,' p. 124.

If there is any conclusion at all justified by the discrepant results obtained by the few who have attempted this kind of investigation, it is that the influence of weather in producing pneumonia is not to be regarded as paramount, that it must rank as a secondary and not a primary, or even an essential, cause of the affection. This may seem a bald return for much labour; yet it will not be denied that it serves to widen our conception of the disease we are studying, and is in harmony with other facts yet to be considered, of which the general tendency is to show that the importance of certain meteorological conditions in the etiology of pneumonia has been overestimated, or at least that these have been too exclusively regarded to the neglect of other agencies not less obviously concerned.

CHAPTER XVI

ETIOLOGY.—II. INFLUENCES OTHER THAN WEATHER AND CLIMATE

Alcoholism—Sewer-gas poison—Starvation—Mental or bodily fatigue—Influenza—External violence—Contagion—Obscure origin.

In considering the causes provocative of pneumonia other than those connected with bodily exposure and climate changes, we have still to discriminate, as far as possible, between causes that of themselves suffice to produce the disease, and those that in varying degrees predispose the body to its attacks. Even in the most characteristic examples of chill pneumonia following some notable exposure, and suddenly interrupting the course of health, few will doubt that there exists some underlying cause besides this obvious one—chill. Nevertheless, the body, exposure, and the pneumonia attack stand to each other in the relation of cause and effect. Had the individual not been so exposed he would not so have suffered.

Is it otherwise with such associations of pneumonia as alcoholic poisoning, privation from food, exposure to noxious emanations, nervous shock, or bodily injury? These, as we have seen, predispose to the affection; do they ever of themselves provoke it? Take, for example, the case of *alcohol.* It is a familiar experience that patients under treatment for delirium tremens will sometimes (often insidiously, and even without discovery during life) develop pneumonia indistinguishable, anatomically, from that which has its chief cause in chill. In some instances of the kind, all risk of exposure has been guarded against, temperature and weather have been equable, and the patient has been kept in bed; and yet the pneumonia comes.[1] It seems as reasonable to suppose in such case that it is the direct result of alcoholic poisoning as to make the like supposition in the parallel case of chill.

Again, there is evidence that *sewer-gas poisoning* not merely predisposes to pneumonia but causes it. As in the case of alcohol, there may have been no weather exposure at all, only exposure to sewer gas; while not one person merely, but several members of a family, are attacked, so that there can be no reasonable doubt that the common cause is with them in the house. Of this form of pneumonia numerous examples might be given. An outbreak at a boys' school at East Sheen, where five persons were attacked, and one died, may serve as a specimen.[2]

On March 14, 1874, in spite of protest on the part of the school, the parish sewer opposite the house was opened for the purpose of ventilation. The ventilator was placed in the high road, and provided with the supposed protection of a charcoal filter. On the 20th an unusually high tide for a time submerged the mouth of the sewer, and the sewer gases forced their way through the ventilator, so that, owing to the odour, it was necessary to close the windows overlooking it. The day following, a boy, whose bedroom was in that part of the house, was taken ill of pneumonia, and the same evening two other boys. Two servants also were attacked, of whom one died. With the closing of the sewer the evil ceased. The house was well drained, and had always been remarkably healthy.

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[1] In 13 examples of pneumonia arising from alcohol, quoted in the 'Collective Investigation Report,' only 3 were subjected to exposure. Of the 10 patients whose pneumonia was presumably due to alcohol alone, all were young or middle-aged, and all but 2 died (*loc. cit.* p. 60).

[2] See 'Coll. Invest. Report,' pp. 7, 58, 60, where other examples of pythogenic origin are quoted.

Cases very similar to this are to be met with in the 'Collective Investigation Report.' Mr. Corbett, of Surbiton, for example, relates the occurrence of pneumonia in three persons (1025–7) in the same house and at one time, owing to a precisely similar cause. The other examples in the report (about nineteen in all) are connected with offensive smells, as from privies and pig-styes (*e.g.* Dr. Raven's report of four children suffering together from that cause, 460–3), or from working in drains and foul sinks. In many of these the earliest symptom is violent vomiting.

In some instances, however, the source of the pneumonia is less obvious, and only discovered after careful search. Thus, Dr. Gooch writes of an epidemic at Eton, June 5, 1883 ('Collective Investigation Record,' vol. i. p. 104). 'The first case was that of the butler, who was taken ill with acute double pneumonia and nearly died. A few days after two boys had had sore throats, and another lad an attack of pleuro-pneumonia. Two days after several other cases of sore throat, and two or three of diarrhœa, occurred in the house, and then another boy was seized with acute double pneumonia, which ran a severe course. At the same time a third boy in another boarding house was attacked with pneumonia, but though boarding elsewhere, he was often at lessons in the house first mentioned. All the patients recovered, but there were twenty inmates of the house more or less ill during a period of about three weeks.'

'We examined,' writes Dr. Gooch, 'the water and milk supply, also the drainage, which was imagined to have been in perfect order, and we ultimately discovered that the trap from the sink in the butler's pantry was defective, and through it sewer gases were escaping into the house. In this pantry the butler slept, and the pupils' room and boys' room are not far distant from it. I think there can be no doubt that all this illness was caused by that defect in the drain mentioned, for there was no epidemic of scarlatina, or any other illness, at the time in the neighbourhood.'

Among the most recent contributors to this subject is Dr. Townsend, of Boston, Mass.[1] This physician, regarding pneumonia as 'an infectious disease, although not contagious or directly communicable from patient to patient,' directs special attention to the frequent concurrence of two or more cases of pneumonia in the same house, the common cause of this invasion being obvious defects in drainage. Out of 643 cases, referring to a particular district between the years 1882–8, 487 occurred in separate houses, and 156 two or more in a house. The mode of distribution is described as follows :—

In one instance seven cases occurred in one court.
In one instance five cases occurred in one house.
In four instances four cases occurred in one house.
In fourteen instances three cases occurred in one house.
In forty-three instances two cases occurred in one house.

Filthy conditions of dwelling characterised the patients here enumerated. One striking illustration may be given where three cases occurred at the same time in the same house, other three happening at about the same time in two neighbouring houses. The three houses concerned were adjoining, all overcrowded and in filthy condition, sinks not trapped, water-closets foul, &c. &c. Six cases of pneumonia occurred in these three houses within five months. In one instance, where three children living in these circumstances were attacked, one of them 'had two relapses, or, we might say, three distinct attacks, in which the right lower lobe, the left lower lobe, and the right upper lobe of the lungs were successively involved.' Further illustrations are given by the author, tending to prove the pythogenic origin of all the cases, and some particulars are added as to the character of individual attacks. Thus, on the day the foul cellar flooring of a small house was disturbed with a view to removal, the earth being saturated with sewage owing to broken drain pipes, a child of the family was attacked with acute lobar pneumonia, which ran a typical course, ending by crisis on the eighth day. A brother living in the same room was similarly attacked the day of the crisis in his sister. He also passed through a normal course of the disease, crisis occurring on the eighth day. The conclusion Dr. Townsend would draw from the facts is, that 'pneumonia is endemic in Boston, and that its growth and propagation are favoured by insanitary conditions.'

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[1] 'Boston Medical and Surgical Journal,' March 27, 1890.

The following is a fairly illustrative case out of many that might be quoted from our own experience in this matter.

Amy B., aged 14, the youngest of a family of six children, was admitted into the Middlesex Hospital on January 11, 1883. She had always been rather delicate, but does not appear to have had before any serious illness.

Her illness began with a pain in the side, increased on taking a deep breath, and on coughing, which had come on at the same time.

It appears that she was living in some penury, having insufficient food, and dwelling with three other members of the family in an alley. She had lately, too, been much exposed to inclement weather, often getting wet through. Two others of the family were taken ill with much the same symptoms before this patient was attacked. One had been ill for a fortnight and was now attending a hospital as out-patient, having been an inmate. The other had been ailing for four days. These illnesses were attributed to exposure to bad odours from an old brick drain which was being replaced by a piping in front of the house.

State on Admission.—The patient is an ill-nourished girl. She has a depressed aspect, the cheeks are flushed, *alæ nasi* working; tongue thickly coated; the skin is hot and dry. She has a troublesome cough.

The chest is resonant on percussion, except at the base of the right lung, from the scapula downwards; here the note is dull, and the breathing (elsewhere harsh and vesicular) of tubular quality, with some fine crepitation and sonorous rhonchus. The abdomen is not tumid, there are no rose spots; the margin of the spleen is not palpable, but the area of splenic dulness is slightly increased. The urine shows a notable diminution in chlorides, sp. gr. 1023, no albumen. T. 102·6.

Cold compresses were applied to the right side of chest, and a mixture containing ammonia prescribed.

During the next day the temperature reached 104·4, falling in the evening to 102·4; P. 120; R. 52.

January 13.—Bowels open four times; motions pale and flaky. More dyspnœa, R. 52. Pain in right side of chest; crepitation audible over lower half of right chest, but tubular breathing limited to a small area in vicinity of angle of scapula. Tongue thickly coated; abdomen natural; T. about 103°.

January 14.—No apparent change. Pulse smaller, and more stimulant ordered; temperature mostly about 103·6.

January 15.—T. at 10 A.M. 98·4; P. 100; R. 52. Passed a better night. She looks brighter and better. Breathing shallow; cough frequent and hacking. Impaired resonance now limited to lower third of right back and axilla. The breath sounds still high-pitched, blowing and bronchial just below angle of scapula, where there is abundant crepitation, which is also audible at base in front. Tongue moist, but thickly coated, except at tip and margins. Temperature throughout the day did not exceed 99·4.

January 16.—Continues to improve; T. 99° to 97·6.

January 17.—Cough less troublesome. T. 99° to 97·8; P. 96; R. 36.

January 18.—T. 97·8 to 99°; P. 76; R. 32. No cough; pulse small.

January 19.—Good resonance over right front and axilla. At back dulness at extreme base, and breathing still blowing at angle of scapula, where vocal resonance increased. Some crackling râles in this region and a few rhonchi over both lungs. Harsh breathing over left lung.

The patient continued to improve, but it was some time before the dulness entirely disappeared from the right posterior base.

There was no recurrence of pyrexia, and although for many days rhonchi were audible over the lungs, they had entirely disappeared by February 12. She left the hospital on February 21.

This case was attributed to drain poisoning, and the fact that two other members of the family suffered at the same time is strong evidence in support of this view. The pyrexia terminated by a distinct crisis on January 15, the fifth day after her admission into the hospital, and the physical signs from that date began to clear up, although it was some time before the lungs were entirely free.

The percentage of mortality of pneumonia having this origin would not appear to be high, and excepting a proportion of cases already alluded to (Chapter X, p. 197), its clinical features are not peculiar.

The causal relationship of *starvation* stands on a similar footing with alcohol and sewer-gas poisoning. Deprivation from food, as we have shown, is often associated with pneumonia—Does it ever cause it? Want of food is apt to concur with bodily exposure, and not seldom with alcoholic excess. In such company its individual share in producing pneumonia in a given case cannot of course be known. But starvation is sometimes met with apart from drunkenness, and in climates where the risk of exposure is comparatively slight. Yet still pneumonia is a frequent mode of death with the starved. Thus, in the Madras Famine of 1877, out of 220 persons dying of want of food, 30 died of acute, and 16 of chronic pneumonia, making 46 in all, or rather over 1 in 5.[1] We have nothing to add, however, in this place to what has already been said (Chapter X, p. 193) of the connection between poverty and starvation, and a form of pneumonia which is rapidly fatal. Considering the comparative rarity of this form, notwithstanding the extreme poverty of many patients brought

[1] See 'Madras Government Medical Reports,' 1878.

into hospitals, we think it doubtful that privation of food is of itself a cause of that disease in the same sense as either acute alcoholism or sewer poisoning.

Of other asserted causes of pneumonia, not provocatives merely, we have also to speak with caution, carefully separating the disease now in discussion from those forms of hypostatic consolidation and congestion of lung so commonly met with in lingering death. There is evidence, however, to support the belief that *fatigue*, *mental or bodily*, *the depression of calamity*, *or bereavement*, whatever, in a word, relaxes the frame and subdues the spirit, favours the advent of pneumonia, and even alone may suffice to produce it. It is vain to inquire in what manner or combination the event comes about; what is certain is, that unless we admit causes such as these, a number of examples of pneumonia will go unexplained, examples, too, which more often than the others have a fatal issue. A man is excited and maltreated by an election mob; or he is prostrated by the loss of wife or child; or some prolonged physical exertion of a painful sort has taken the 'life' or the 'spirit' out of him. At first he is not actively ill, but merely 'depressed,' 'cast down,' 'not himself.' Presently pneumonia comes, and with it, if not of it, the man dies. In such cases—and they are taken from life—the lung inflammation is the one definite tangible feature in the illness, and serves to give it a name, while the mental shock is the one fact in the patient's history that seems to give it a reason. Yet when all the circumstances are taken into account, it is apparent that the lung inflammation really depends on an antecedent nervous change determining both its advent and its fatality, but of which, histologically, we know nothing. Thus, by degrees, pneumonia recedes from view in its simple form as a local inflammation due, like pleurisy, to some accident of exposure, and comes to occupy but a subordinate place in a series of phenomena of which it is the latest and, in physical respects only, the most obvious. Apart from its pathological interest, pneumonia of this origin deserves notice owing to its high mortality, a feature that it shares with the alcoholic form of the disease. In fourteen examples supposed to originate in this way, as many as nine died, a proportion the more remarkable on analysis, whence it appears that all were temperate and sufficiently fed (two being total abstainers), and but one advanced in life.[1]

In this place we may mention the remarkable predisposition to pneumonia, both lobar and lobular, noticeable just now (1890) —in the prevailing epidemic of so-called *influenza*. It is but an illustration of what we see elsewhere to find that individuals, weakened by an illness characterised by remarkable loss of bodily power and severe mental depression, are often the subjects of pneumonia of exceptional severity. But the frequency with which lung inflammation follows influenza is too great to be accounted for by predisposition merely, and argues an affinity between the two as yet, in our opinion, unexplained. At the present time, as will be shown in the next chapter, the agency of particular microbes in the production of the disease is a vexed question, and nothing has yet been advanced to prove that pneumonia and influenza own a common origin of this kind.[2]

It is of more practical importance to note the extreme gravity of pneumonia as a sequel to, or accompaniment of, the influenza of 1889–90. It has been recently fatal in two cases under our observation, namely, a man of twenty and a woman of fifty-two. In the first patient bronchitis was present, but the physical signs at the base of the right lung seemed to indicate a lobar consolidation; the pulse was 115 and temperature did not much exceed 103°. On the 12th day the latter began to descend, pulse and respiration much increasing. There was no albuminuria. As the man became collapsed, respirations were nearly 70 and pulse 128, while the temperature had fallen to 97°. He died on the 14th day. No post-mortem was allowed.

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[1] 'Coll. Invest. Record,' p. 59.

[2] Unless the detection by Weichselbaum and others of the *diplococcus pneumoniæ* in the catarrhal mucus of influenza cases is to be assigned more importance than in our opinion it merits.

In the second case, the woman's household had suffered influenza, and she had been up six nights consecutively, nursing her son in pneumonia, following that affection. This terminated six days before the mother's admission, when she herself, already much exhausted, showed symptoms of pneumonia. She died on the 9th day of illness, temperature but slightly raised until just before death, when it mounted to 105°. Here there was extensive hepatisation of both lungs, the right being grey; the other organs were healthy.[1]

Owing partly to the great attention excited by a widespread epidemic, and partly to there being much in common between the initial symptoms of influenza and those of pneumonia up to the time of development of physical signs in the latter, there can be little doubt that many examples of pneumonia were ascribed to this source upon insufficient grounds. A case of our own, quoted in Chapter X, p. 204, may be of this kind. It is still certain that the epidemic prevalent at the time of writing confers an exceptional liability to acute bronchitis, broncho-pneumonia, and lobar pneumonia, probably in that order of frequency; and that in this connection these diseases are of exceptional severity, though seldom fatal, except to old people and those of weakly constitution.[1]

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[1] Some observations by Drs. Robertson and Elkins on an Epidemic of Influenza (140 cases) at Morningside Lunatic Asylum, Edinburgh, accord with our own much smaller experience of that disease in this relation. 'Pneumonia,' they say, 'was not of the pure croupous variety, bronchitis always preceded. The condensation was gradual, and crepitation always of medium coarse variety; the sputum frothy, streaked with blood and *never rust coloured.*' In 10 fatal cases (all males) the main cause of death was pneumonia, greatly assisted by the very weak general health of the patients attacked. Six of the 10 had general paralysis, 3 were over 70, and 1 alcoholic and with serious heart disease. No details of post-mortem examinations are given if any were made. 'British Medical Journal,' February 1, 1890, p. 228. The experience gained in the recent epidemic in the United States has been illustrated by papers read at the New York Academy of Medicine in April, 1890, by Drs. Pepper of Philadelphia, Janeway of New York, and Shattach of Boston, upon pneumonia following influenza. Dr. Pepper pointed out the frequency of the association, and considered the pneumonia to be a sequela rather than a complication of influenza. His cases showed an undue prevalence of right-sided pneumonia, of jaundice and albuminuria (see 'Boston Med. and Surg. Journal,' April 24, 1890).

It is no very rare experience to find pneumonia and *bodily injury* in near association, yet the precise nature of the relationship is open to dispute. The question is whether external violence (short of lung laceration) is capable of producing lung inflammation, having all the characters of true lobar pneumonia as regards its course, character, and end.

Grisolle, an excellent authority on matters of fact of this kind, hesitates to reply; he hardly admits the possibility of such an occurrence, and uses *à priori* arguments against it; yet he quotes examples of pneumonia resulting from heavy lifting. Dr. Frederick Roberts places 'injury to the chest' among 'the exciting causes' of pneumonia.[2] Dr. Bristowe speaks of 'the direct action of mechanical irritation,' but does not appear to include in this expression external injury.[3] Dr. Wilson Fox[4] says, 'Injuries and blows to the chest are occasionally followed by pneumonia'; yet he admits that 'the mechanism of such cases is obscure,' and the single example which he supplies from his own experience is, as he himself seems to imply, far from conclusive. Of earlier authorities, Sir Thomas Watson dismisses the causes of pneumonia in precisely six lines,[1] and in none of the six does he mention injury. Going further back still, we come to foot-note references copied from author to author, and to which time and tradition have given a sanctity seldom invaded by any close inspection. Amongst these is a case quoted by Andral,[2] where, however, the writer himself remarks that the almost complete absence of fever on the fifth day after the accident (a fall from a height) would have negatived the theory of pneumonia, but for the sputum and the crepitant râle. This was written at a time when physical signs, taken separately, were more absolutely trusted than at present. An impartial reading of the case in question leaves the impression that an interval of five or six days intervened between the fall and the first appearance of pneumonia. Upon that supposition the 'amélioration étonnante' which befell five days later would correspond with what we now call crisis, and the accident itself would be too distant to be safely reckoned as the immediate cause of the pneumonia.

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[1] As an example amongst many of the rare fatality of influenza in this as in former epidemics, we may here quote some statistics from the University Press, Oxford. Two hundred and thirty-six of the employés were attacked, 211 men and boys, and 25 women and girls. Not one died. Horace Harte, Controller, in letter to 'Times,' February 7, 1890.

[2] Roberts, p. 399, fifth edition.

[3] Bristowe, p. 403, fifth edition.

[4] 'Reynolds's System,' vol. iii. p. 615.

The following narrative[3] well illustrates the difficulty of determining the precise significance of bodily injury when occurring in near connection of time with true pneumonia. It is abridged from very full and careful notes taken by Dr. John Thomson, of Edinburgh, at that time house-physician.

Charles T., aged 11, an ill-nourished, pigeon-chested child, dirty and neglected, was admitted to the Alexandra Ward of the Hospital for Sick Children on the morning of January 9.

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[1] Watson's 'Lectures,' vol. ii. p. 89, third edition.

[2] 'Clinique Médicale,' tome iii. p. 293.

[3] See also a case (with comments) published in the 'Lancet,' by Mr. F. W. Jollye, June 16, 1888, and two cases reported by Dr. Foss, May 31, 1884.

He was much collapsed, the face pale, and extremities cold and clammy. The breathing was very shallow and rapid; respiration 50; pulse 176, regular and very compressible; temperature 103°. Lips and tongue dry and cracked. Though drowsy and indifferent he could yet answer questions, and when moved gave sign of pain in the right chest. There was no tenderness, however, at this place, and no bruising anywhere. When left undisturbed the breathing became slightly stertorous. On physical examination, a patch of impaired resonance was made out below the right scapula, and here the breathing was faint and bronchial, and the vocal fremitus exaggerated. There were rhonchal and sibilant sounds generally throughout the chest, but nowhere crepitation. From such physical and general signs it was concluded that the child had pneumonia in an early stage; but so grave was the prostration and so enduring, notwithstanding free stimulation, as to suggest that there must be something more, as yet unseen, which would be presently revealed. The history obtained, partly from the patient and partly from his mother, an inaccurate, unintelligent woman, was to the following effect:—

On the day before admission the boy went to school as usual. Between three and four in the afternoon he came home complaining that while on his way to meet his father, a big boy who happened to be passing had hit him a severe blow with his fist on the right side of the chest. He walked home with difficulty, perhaps a mile, feeling the pain, which movement seemed to aggravate. He had also headache and a sensation of nausea, and it was observed that he would restrain cough owing to the pain it gave him. On reaching home he rapidly grew worse, and after a restless and feverish night was brought to the hospital early next day in the condition already described. On a rough calculation some fourteen hours may have elapsed between the time of the blow and the time of admission, but so far as could be gathered only a very short interval between the assault and the appearance of serious symptoms. The apparent origin of the symptoms was sufficiently remarkable—especially, as will be presently seen, when these had fully developed—to make strict inquiry desirable as to the child's previous health; and accordingly, Dr. Thomson, by dint of much questioning, succeeded in eliciting from the mother, on her second visit, some important additions to her original statement. These were to the effect that the boy had 'shivered a little two days before the blow,' that he had complained of his throat, and that the mother, who was far from being an anxious observer, had noticed that he 'looked ill,' and told him to remain at home, and.

finally, that on January 5, three days before the blow, he had been out in the snow all day and got wet through. The after-progress and termination of the case may be dismissed in a few words. The day after admission all the characteristic signs of consolidated lung became apparent as regards the upper third of the right side. Prostration continued ; the temperature on the third day reaching 104°. Then came a critical fall of 6° on the fifth day from admission, and along with it all serious symptoms vanished, and a few days later the boy was convalescent. This crisis, it should be observed, corresponded with the sixth day of illness reckoning from the blow, with the eighth reckoning from the shivering, and with the ninth reckoning from the wetting.

This case would seem at first sight to give an affirmative answer to the question, Can injury cause pneumonia? It is not easy to find another so explicit. At the time of the blow the boy was walking the street. It causes violent pain, and he gets home with difficulty. Dyspnœa, fever, and pleural 'stitch' set in almost immediately, and a few hours later, when medically examined, the physical signs of pneumonia are discovered. The proof seems strong that a blow on the chest *is* a possible cause of that disease.

But further inquiry gave a somewhat different colour to the facts. It was ascertained that four days before the blow the boy had got wet through, and that he afterwards shivered and looked ill. Now, pneumonia from exposure to wet is common, while pneumonia from a blow in the chest is, at best, very rare. So considering, and with the actual sequence of events before us, the probability is that pneumonia had already begun in the right lung at the time when he was dealt a blow from a boy's fist just over the seat of it. That violence of this sort directed against a deformed and yielding chest should produce a temporary collapse is, upon this hypothesis, natural enough. As this collapse gradually yields, the proper physical signs of pneumonia appear in typical completeness.

A similar explanation would seem to apply not only to external violence, but also to violent exertion on the part of the patient. Grisolle[1] quotes examples where pneumonia was attributed to the lifting of heavy weights. 'A violent pain was immediately felt, and, in a few hours, symptoms of pneumonia declared themselves.' Now, while it is difficult to suppose that extra muscular exertion can give origin to this disease, it is quite probable that it might serve to discover it. Such extra inspiratory effort as heavy lifting implies would be well calculated to *find out* a pneumonia which was just commencing, and as yet, while the patient was at rest, had given little notice. It is thus, as we believe, that a fall or other accident sometimes gets the credit of causing lobar pneumonia in young children. In like manner, other acute affections, such as enteric fever, are sometimes brought to light through the test of some muscular exertion which could not, by any possibility, have had any share in causing them. Thus, in our own experience, a young sweep, carrying his bag of soot one morning, fell heavily under its weight, and was brought to hospital in serious collapse. He was found to be in the second week of enteric fever, of which he died.

But while it may be doubted whether either external violence (short of lung laceration) or over-exertion ever directly excites pneumonia, there is ground for the belief that both one and the other may give occasion for it. For there is a form of the disease, as we have seen, of which the predisposing cause—sometimes so far as appears the sole cause—is deterioration of vital energy ; a bodily condition to which at present we can apply no more precise expression. Whatever lowers vitality invites pneumonia. In virtue of that principle, severe bodily injury, as well as over-fatigue, alcoholic excess, destitution, or even mental anxiety, may weaken the power of resisting the disease and make its invasion the easier. Pneumonia of this origin needs the more to be recognised, inasmuch as it is a grave and often fatal disorder, with quite a different outlook from the pneumonia of exposure. To such category would belong the case just quoted from Andral, possibly also some examples of railway and other accident, which might be quoted, where individuals debilitated by an injury, but apparently on their way to recovery, die quite unexpectedly by the intervention of a pneumonia of such limited area as to seem at first a mere trifle.[1] Considerations of this sort make us properly cautious in accepting injury or overstrain as of itself the efficient cause of pneumonia. Granting that individuals may be up and about in every stage of that disease,[2] granting also that the early symptoms of pneumonia—dyspnœa, pain on deep inspiration, and general muscular weakness—are precisely those which extra exertion or a blow directed against the chest or diaphragm would call into activity and prominence, and it is inevitable amongst a hardworking and boisterous race that accidents of this sort should from time to time call attention to the disease, and appear in the light of causes of the symptoms which they precede.

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[1] 'De la Pneumonie,' p. 144.

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[1] See 'British Med. Journ.' 1879, vol. i. p. 100. The point is of interest forensically. It has been raised in cases where compensation or the payment of a policy of insurance is claimed owing to fatal pneumonia following railway or other accident. For example, a man is shaken owing to a train coming to an abrupt stop, or his shoulder is dislocated by falling on the platform. After an interval of a few weeks pneumonia sets in and the patient dies owing, as it is alleged, to the accident. In judging of any such case it is of course necessary, in the first place, to make sure that true lobar pneumonia was the disease suffered from and the immediate cause of death. This ascertained, the tenour of medical evidence would seem to depend upon the individual's condition in the interval between accident and death. If it could be shown that the bodily or mental health had suffered, and that the normal strength and spirits had never returned, then, however indefinite the symptoms of such failure and with an interval of some weeks, a medical witness would not be justified, in our opinion, in maintaining that accident and pneumonia were not in relation of cause and effect. On the other hand, if it could be proved that the injury was followed by no such evil consequence, and that at the onset of pneumonia the patient was in his usual health, then, considering the numerous modes of origin of the disease, its commonness, and the unquestioned rarity, to say the least, of what is called 'traumatic pneumonia,' it would be in the highest degree improbable that an injury of the kind contemplated would be connected as cause or part cause of the subsequent illness and death. Still stronger, perhaps, is the case (lately submitted to arbitration) where pneumonia occurs shortly after an accident and recovers, but after a while the individual dies of a different disease. For in that case the mere fact of recovery affords some proof of the man's vigour, while it is well known that the true lobar pneumonia of which we are speaking rarely entails any after consequences prejudicial to life.

[2] The number of instances in London alone where people are found in the streets dead or dying of pneumonia in an advanced stage, yet which has never been suspected, is not small. Hospital practice, though it gets a share of these cases, does not adequately represent their frequency. For

There is a further question, which we have here expressly excluded, regarding injuries causing laceration of lung tissue with or without fraction of rib. Can such laceration, however caused, set up true lobar pneumonia? It is for surgeons to say ; certainly such an event is rare.[1] The pulmonary inflammation of traumatic origin is usually limited in extent to the margin of lung surrounding the laceration, blood contents and inflammatory products occupying the alveoli in this situation. The course and history of such inflammation bears no resemblance whatever to what happens in pneumonia.

In like manner the *application of irritants* to the lung, by inhalation or otherwise, produces no more than a limited

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that we must go to Poor-law infirmaries and coroners' inquests. A working man of middle age, well proportioned and developed as to his frame and muscles, who had been feeling out of sorts for some days, was yet able to take active part in a meeting for political discussion ending with whisky and water. On his way home he met with a fatal accident, falling headlong down some stone steps and fracturing his skull. Post-mortem, part of his right lung was found to be solid, and on section and microscopic examination it proved to be a specimen of typical pneumonia, probably in course of resolution. Section of the lung is figured on p. 72.

[1] Dr. Thos. Barlow, physician to University College Hospital, has been good enough to communicate a case of his own which is possibly of this sort. A young stonemason had a heavy stone slab fall on his chest, knocking him down with a very severe blow, yet without fracture of ribs. 'He was taken home, and within a day or two developed a typical apex pneumonia. This resolved, but after resolution there was a curious hectic temperature, a condition lasting a considerable time, the morning temperature being normal and the patient meanwhile feeling well and ultimately recovering perfectly.' Dr. Barlow adds, 'At the time I did not think much of the immediately antecedent injury, but I remember the fact very definitely.'

alveolar inflammation. Thus Gendrin from inhalation of chlorine obtained exudation into the alveoli, giving the naked-eye appearance of small white nodules, and Bretonneau got similar results from the fumes of hydrochloric acid. Cruveilhier, injecting mercury into the bronchi, with the object of exciting tubercle, produced little zones of pus surrounding each globule of mercury. Other irritants, such as hot steam, have given like results. From the injection of blood into the trachea Sommerbrodt produced only epithelial desquamation, while perchloride of iron gave rise to fibrinous exudation with the production of leucocytes.[1] The pneumonic nodules are often apparently preceded by collapse, and from reddish become yellowish spots, eventually, in some instances, softening into small abscesses. These results, obtained from experiment upon animals, hardly concern us here, and, in so far as they remind us at all of the processes of disease, are akin to lobular and not lobar pneumonia.

The final conclusion in regard to the alleged traumatic origin of the disease seems to be this. Lobar pneumonia does undoubtedly sometimes follow injury, either immediately or with a short interval. In some instances this sequence is accidental, the inflammation not being caused by the injury, but only brought to light through its agency. In others there is a real but indirect relationship ; the pneumonia arises, that is to say, not from any harm done immediately to the lung, but from the nervous shock which bodily injury, whether of the chest or elsewhere, is apt to produce, and which, in common with fatigue, destitution, and alcoholic excess, predisposes the individual to such attacks. We have here, in fact, only a further illustration of the occurrence of true lobar pneumonia of definite duration and orderly course, not as the consequence of exposure, but as a local expression of some obscure nervous change.

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[1] Quoted from Wilson Fox, ' An Atlas of the Pathological Anatomy of the Lungs,' 1888, p. 35.

Some observers have asserted, especially of late years, that pneumonia is not only epidemic but also *infectious*, meaning by that term that in certain epidemics[1] it is conveyed directly from person to person, but not, as we understand, that it is communicable through the intervention of a third person not himself affected, or that this alleged communicability is other than rare and exceptional. In the 'Collective Investigation Report' there are thirty examples of supposed infectious pneumonia, eight of them fatal; or, if ambiguous cases be rejected, there are twenty such examples, five of them fatal. In dealing with such cases it is necessary in the first place rigidly to exclude all *à priori* considerations based upon the presence of specific organisms. We are not asking whether pneumonia ought to be infectious, but seeking to ascertain whether the fact is so.

Moreover, it is obvious to remark that any disease whatever in its epidemic prevalence is certain to suggest infection, whether really infectious or not. The spread of pneumonia through a district or street or household is no evidence of infection; it is evidence only of epidemic prevalence in that particular area; and undoubtedly pneumonia, like other affections of the respiratory organs—like bronchitis, catarrh, and coryza—is apt to occur in groups and to seem as though caught from some earlier sufferer. What is required for proof of real conveyance is this, that the affection should spread when imported into a new place hitherto free of it; and that this should occur with sufficient frequency to override accidental coincidence. On the other hand, it is no valid argument against the possible yet exceptional transmission of pneumonia from person to person, a disease so various in its origin and attributes, to urge that infectibility is not one of the ordinary characters of the disease. Pneumonia, whether as such or as the expression of some specific poison, itself unrecognised, may be contagious in exceptional cases without necessarily exhibiting that property in our hospital wards. Nothing seems more certain than that the disease, as we commonly see it in London, is incommunicable. We are familiar with it in many forms; in seasons of prevalence it is not uncommon to see two or three or four sufferers occupying the same ward. Yet, so far as we can learn, there is no distinct example of the affection even seeming to spread from patient to patient in any London hospital. That pneumonia is not commonly contagious seems quite certain; but is it ever contagious? It is a question of evidence, and, according to some observers, the answer must be in the affirmative. Thus, Dr. Patchett relates the following narrative[1]:—

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[1] The infectious character is nowhere asserted, so far as we know, as belonging to pneumonia always. The contention is that 'epidemics of pneumonia of an infectious character occur at rare intervals.' Bruce, 'Brit. Med. Journal,' May 15, 1887.

A family of five elderly people, four brothers and a sister, lived together on a steep hillside, with good sanitary surroundings and apart from any known epidemic or septic influence; the men well-to-do farmers. On January 13, 1876, James, the eldest, was taken ill and died in six days with typical pneumonia. The day before his death, John, the next in age, had a rigor, and three days after his brother he also died of pneumonia. The day after John's death the two remaining brothers both suddenly got pneumonia, and both speedily died of it. There was now but the sister left; she had nursed her brothers throughout and 'kept her health remarkably well.' But the day after the death of the two last she also was seized with pneumonia of the right lung and died in three days. Thus, in less than a fortnight, the entire family were swept away by a disease which the narrator describes as 'typical pneumonia.'

The second illustration is by Dr. Daly,[2] and is hardly less striking:—

A child of the family concerned was ill and feverish, but no very definite symptoms were noted, and he soon recovered. Nine days later, however, two other children were seized with acute pneumonia. On the fourth day of their illness the mother, who was a constant attendant in the sick room, took the same disease, as well as the youngest boy. With four undoubted cases of pneumonia in the same house, within a few days, it occurred to Dr. Daly to examine the child who first of all had shown signs of illness. He satisfied himself that this child too had suffered from pneumonia. In the later part of her illness the mother of the lady came to nurse her daughter. The latter died, and six days later the mother also died, and of her daughter's disease. The sanitary condition of the house was most carefully investigated, and no fault could be discovered; there was no epidemic prevalent, and none of the patients had been out or otherwise exposed. Further, it was noticeable that those members of the family who were frequently together were all attacked, the rest escaped.

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[1] 'Lancet,' 1882, vol. i. p. 305.

[2] *Ibid.* 1881, vol. ii. p. 824.

In some instances, no doubt, the theory of contagion is invoked in default of any other available explanation. This is well shown in a report furnished by Dr. Gilbert Child, medical officer of health, of an epidemic of pneumonia at Long Handborough, Oxon, April 1878:[1]

A remarkable outbreak of a disease not usually recognised as belonging to the zymotic class occurred at Long Handborough in the month of April. This was an epidemic of pneumonia, which carried off no less than eight persons in this village between April 6 and April 27. In all cases the victims were adults—the youngest twenty-two years of age, the oldest eighty-seven, and the remaining six between the ages of thirty-eight and sixty; and in every case the progress of the disease was rapid, death occurring in most cases within a week of the attack. One remarkable circumstance in connection with the outbreak was that a woman living at Witney (about five miles distant), a near relation of one of the persons who died at Handborough, walked over to the latter place to attend the funeral, remained there for the night, sleeping in the bed from which the deceased person had been removed. She returned to Witney on the following day, sickened with the same disease, and died within a few days. This might seem to suggest that the complaint was infectious, but the conclusion is by no means certain, as it is clear that if the disease depended on any local conditions, the visitor had in this case subjected herself, for the time, to almost the precise combination of them which prevailed more permanently among the inhabitants of Handborough. At the same time I must admit that I am quite unable to assign any local conditions as the cause of this outbreak. A few similar ones have been recorded elsewhere, amongst which I may mention one at Holmwood, in Surrey, about a year before the present one ; another of which I have been informed at a large school in Berkshire several years ago ; and one at Cowley, near Oxford, almost simultaneously with that on which I am now reporting. At present I do not know of any satisfactory explanation which has been suggested for such outbreaks.

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[1] 'Coll. Invest. Report,' vol. i. p. 105.

The subject of contagion is of such interest just now in connection with the pathology of pneumonia, that we may refer to some further evidence in reference to it derived from foreign sources.[1]

A. Müller[2] gives a striking instance in a household at Garvenhausen in November 1873 :—

The first to be attacked was the mother (A) aged fifty-eight, on November 25 ; on December 6 the father (B), aged sixty-four ; on the 7th the son (C), aged eighteen, and about the same date a daughter (D), who did not live with her parents, but who had been attending on her mother during her illness, and a grandchild (E), five years of age, who took ill on December 10. In all the cases but E there was a marked initial rigor, and in all but A the attack was marked by gastric symptoms, C and D especially suffering severely from vomiting. There was great prostration in all ; the parents A and B being also delirious and having an irregular, feeble pulse. The physical signs of pneumonia were marked and the expectoration characteristic. The inflammation involved the left upper lobe in A and E, the right upper lobe in B, and right lower lobe in C. All recovered by crisis, which took place on the ninth, seventh, and fifth days respectively.

In Wagner's clinic at Leipsic in the summer of 1880, Heitzel[3] met with the cases of three sisters, aged seven, nine, and eleven years, who fell ill with croupous pneumonia consecutively. In each case the disease ran a typical course and terminated favourably. There had been no exposure to cold; but it is interesting as suggestive of a family tendency to note that the eldest sister, who was the first to be attacked, had thrice previously suffered from pneumonia.

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[1] See 'Coll. Invest. Report,' vol. ii.

[2] 'Deutsch. Archiv f. klin. Med.' 1878, xxi. p. 127.

[3] 'Ueber infectiöse Pneumonie,' Leipzig, 1883.

Ritter[1] gives an instance of five persons dwelling in the same house being attacked within five days (March 13 to 18, 1879), and two others, who had visited the house, later falling ill. Of these seven cases three proved fatal, the disease being of typhoid type, with splenic swelling, &c.

Bielenski[2] records the incidence of pneumonia upon nine out of ten inhabitants in one house, attacked within two weeks of each other, and all recovering. Here, moreover, there was not only overcrowding, but the house lay near a graveyard.

Mendelsohn[3] gives other illustrations, notably one in which a convalescent from typhoid fever died from pneumonia contracted, it was believed, by his transference to a bed just previously occupied by a case of the latter disease.

An account of three cases of pneumonia admitted into the Lyons Hospital in six days (December 16–22, 1888) is given by M. Proby.[4] The patients were lads employed in a bakery, and they had in turn occupied the same bed. It appeared that a child of the master baker had just recovered from a 'fluxion de poitrine' when the first lad fell ill.

A remarkable history of apparently contagious pneumonia has lately been recorded by Dr. F. Mosler of Greifswald.[5] The victims were all members of the same family, four in number, and all were attacked during the latter half of January, 1889.

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[1] 'Deutsch. Archiv. f. klin. Med.' 1880, xxv. p. 53.

[2] Quoted by Mendelsohn.

[3] 'Zeitsch. f. klin. Med.' vii. 1883, p. 178.

[4] 'Lyon Médical,' 1889, No 40.

[5] 'Deutsch. med. Woch.' 1889, Nos. 13 and 14.

The first to fall ill was the father, sixty-four years of age, who died on the 22nd, the fifth day of his illness; and the same day his wife, who had nursed him, was attacked, and she too died after five days' illness. Their son, a strong man thirty years of age, not living with his parents, but constantly visiting them during their illness, was attacked on the 26th, and in him, too, the disease proved fatal on the twelfth day. His sister, in service at Arendsee, near Stralsund, who had come home to visit her parents from the 22nd to the 26th, was, on her return to Arendsee, also attacked with pneumonia on the 29th. She was admitted into Greifswald hospital and recovered. Dr. Mosler acquits the dwelling of the parents of any share in the causation of the disease; the two rooms they inhabited were clean and well-ventilated, the house was dry, and none of its inmates had been ill for the past five years. He believed that the father—a workman—must have contracted pneumonia outside, and that through him the disease, which was of a malignant type, was communicated to his wife and children, probably by the sputa. Full details are given of the post-mortem examination in the case of the son, and the hæmorrhagic lobular character of the pneumonia noted. A form of bacillus, differing from those mostly described in pneumonia, was obtained from the blood in this case, and also from a puncture into the pneumonic lung of the daughter.

Further examples might easily be quoted [1] where pneumonia has appeared to spread by contagion, but for the most part they are reported in summary fashion, without sufficient detail to be of much value. It goes for very little, however striking to relatives, that an individual attending on pneumonia, whether as nurse or doctor, shortly after suffers in the same way. In the absence of further information, indeed, as to season, dwelling, and epidemic prevalence, it goes for nothing at all.

Finally, and in illustration rather of what is asserted than of what, in our opinion, can be proved, we may quote the conclusions of Netter[1] as abstracted by H. Barbier in the 'Gazette Médicale,' June 22, 1889. Setting aside all cases of epidemic character, his observations concern healthy individuals who, in presence of patients suffering pneumonia, have been themselves attacked. Two of his quoted examples are those of Daly and Patchett, given in an earlier part of this chapter, and the rest are so similar in all respects to those we have already narrated that it is needless to repeat them.

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[1] For example, 'a farmer took pneumonia and died. A week later his servant took it and went home, where she communicated the same disease to a married sister' (Dr. Wynter Blyth, 'Lancet,' September 18, 1875). Many accounts of so-called contagious pneumonia have doubtful right to that character, owing to the fact that the disease is epidemic at the same time and in the same place (see Chapter XIII, Epidemics, p. 268 *et seq.*).

'What,' he asks, 'are the conditions of contagion? Chiefly constant and prolonged relations with the patients or being in the same bed; next, those in attendance on the patient or staying with him for a short while, even a casual visit to the sick room.'

Furthermore, in the opinion of this writer, pneumonia is communicable through an intermediate agent, whether person or thing. The individual attacked need not have seen or approached the patient. The bedding, the bed itself, or even the furniture, may contain the contagion, which, according to Netter, who quotes many authorities (Mendelsohn, Flindt, Schroeder, &c.) has been shown to be active at the end of fifty days. Yet, happily, it is but slightly diffusible, 'an interval of three beds' (which, it may be remarked, is no very exact or constant measurement) being, so far as at present observed, its utmost range.

It need not be added that this writer goes on to enjoin the strictest antiseptic precautions—isolation, destruction of sputa and bedding, and all the recognised methods of disinfection.

Without venturing any more positive statement, we may at least assert that there is less clinical evidence of the occurrence of pneumonia by contagion than there is for its origin from exposure, from sewer gas, from want of food, even from mental o bodily depression, and, if it be granted that any of these agents may possibly provoke pneumonia, it may be doubted whether it is further necessary, in order to satisfy the facts, to make any large appeal to this origin by contagion. For, on this hypothesis, it follows that, apart from contagion, the mere fact of pneumonia happening in a family brings the liability to suffer in the same way the nearer to the other members of it. This it does by importing the admitted sources of the disease—anxiety, fatigue, and (with the poor) often privation as well. And when death enters this is not all; for while these predisposing causes are still in operation there comes in addition, in many cases, standing bareheaded at the grave-side, involving that very exposure to cold which all admit to be the least equivocal of all the causes of pneumonia.

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[1] 'Arch. de Méd.' 1888.

Even admitting as proved all the modes of origin we have enumerated in this chapter, it must still be added that the source of pneumonia is sometimes altogether unaccountable.[1] Like other acute diseases commonly attributed to chill (like acute articular rheumatism, facial erysipelas, and quinsy), pneumonia occurs sometimes, as it were, spontaneously. Yet if it be considered that until recently the field of inquiry as to the etiology of affections of this class was extremely narrow, it is less matter for surprise that the exciting cause of pneumonia is still sometimes altogether obscure, than that we are in possession to-day of so much solid evidence in proof of its origin from other sources than bodily exposure.

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[1] There is a form of acute lobar pneumonia that is preceded for a length of time, longer or shorter, by ill-defined symptoms of illness, such as loss of appetite, bronchial catarrh, and, in children, vomiting, diarrhœa, and night fever. Presently, and with the suddenness and in the manner of chill pneumonia, comes a rigor or convulsion with pyrexia, and the physical evidence of lung consolidation shortly followed by crisis and recovery. It is observable that this acute attack running its course, as usual, in a few days will lead to a convalescence which is in fact better health than the patient has known since he first vaguely sickened. And the symptoms from first to last, both chronic and acute, seem all of a piece. The sudden lighting up of inflammation within the lung is like the storm that dissipates sultry weather, while the quick restoration to sound health that follows seems to indicate that some morbid material or combination has been destroyed or unloosed in the process. In a similar way do the premonitory symptoms of gout gather to a head, explode, and leave the patient better than they found him. Yet, certainly, pneumonia arising in this way has no near connection with gout, and is, in fact, commonest with children.

CHAPTER XVII

BACTERIOLOGY

The progress of bacteriology—Grounds for a possible bacterial origin of pneumonia—Observations of Klebs and Eberth — Friedländer's research—His 'pneumococcus'—Comparative rarity of this microbe—Fraenkel's 'diplococcus' — Its greater frequency — Researches of Weichselbaum, Talamon, Sternberg, and others—The microbes of normal saliva, and their relation to sputum septicæmia on the one hand, and pneumonia on the other—Presence of the diplococcus in other diseases associated with pneumonia or independently of it—Meningitis, pleuritis, pericarditis, peritonitis, endocarditis, otitis, &c.—Other microbes in pneumonia—Klein's bacillus—General outcome of the inquiry—Conflicting opinions.

It was hardly to be expected that amongst the many diseases which have passed under the scrutiny of the bacteriologist, acute pneumonia should not find a place. Indeed, there are several reasons which would appear to promise great results from this research, as determining the vexed question of its etiology. For, as we have seen, amidst all the varied conditions favouring its development, there has remained room for the revelation of some deeper, underlying cause which might be expected to harmonise many seemingly conflicting facts besides according fully with the phenomena exhibited by its clinical history. The conclusion that in this disease we have to deal with a general rather than a local process has long been suspected. It has been adopted by Flint, Jürgensen, and many other writers, and was the view taken in the first edition of this work. This belief almost of necessity leads to the doctrine of the specificity of pneumonia, and to its inclusion in the category of so-called 'infective disease.' Reserving, however, the full discussion of this subject to the next chapter, it may suffice here to call to mind those leading characteristics which seem to justify further inquiry into the connection of pneumonia with an organised virus.

We have seen how closely the affection approximates in its general features to those of acute specific diseases. We have noted its appearance in epidemic form, its frequent close association with defective hygiene, and in some instances (rare, it is true) its transmission under circumstances highly suggestive of contagion. Thus there seems ample ground for believing that pneumonia may be found to depend essentially upon the operation of agencies similar in kind to those assumed, or proved to give rise to other infective diseases. It remains to be seen to what extent bacteriological research gives independent support to this doctrine, and whether the results, so far obtained, have led us any nearer to the solution of the problem of the nature of the disease.

It will be impossible for us here to give more than a mere outline of the progress of inquiry in this subject, with such conclusions as may seem to be justified by the remarkable series of investigations to which we shall refer.[1] They may, perhaps, be most conveniently dealt with in the order of their prosecution, so as to present a historical record of a research which is undoubtedly still open to further developments.[1]

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[1] We are unable to speak on this question as practical workers in bacteriology, and design no more than, by an impartial recapitulation of the statements of original observers in this field of inquiry, to put the reader in possession of the facts. We have endeavoured to supply our own deficiencies by a careful study of most of the available literature on the subject. Among the chief sources of information we have consulted the following works:—Ziegler, 'Lehrbuch der allg. und spec. path. Anat.' sixth edition, vol. i. Jena, 1889; Klebs, 'Die allgemeine Pathologie,' Jena, 1887; Cornil and Babes, 'Les Bactéries,' second edition, Paris, 1886; C. Fraenkel, 'Grundriss der Bakterienkunde,' Berlin, 1887; Crookshank, 'Manual of Bacteriology,' second edition, London, 1887; Friedländer, 'Virchow's Archiv,' lxxxvii. 1882, and 'Fortschritte der Medicin,' 1883 and 1884 (also in the volume of collected papers 'Micro-organisms

The credit of having been the first to discover micro-organisms in connection with the disease rests with Professor Klebs, who, in this as in so many other diseases, has been a pioneer in bacteriology. Nevertheless, the descriptions given by him do not in many respects accord with those of later observers. He found in the sputa and blood of pneumonic patients certain spherical and mobile organisms, some of which showed transitional forms to a rod shape. He termed them 'monadineæ,' and made cultivations and inoculation experiments, which, however, were not very conclusive. Klebs himself thinks that he found the same organisms as were subsequently described by Friedländer. His observations were in a measure confirmed in 1881 by Eberth, who discovered similar microbes in the pleuritic false membranes of a case of pneumonia, and also by Koch, who detected oval-shaped cocci in the hepatised areas and in the kidney of a case of the same disease following relapsing fever.[2] But these isolated, and in some respects imperfect, observations were lost sight of in the more striking and convincing researches of Friedländer, of which the first publication was made in 1882.[3]

In this paper Friedländer confines himself to describing the microscopical characters of a micro-organism which he

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and Disease,' New Syd. Soc. 1886); Klein, 'Micro-organisms and Disease,' third edition, London, 1886; and for references to recent literature from all quarters, the 'Centralblatt für Bakteriologie,' &c., vols. i. to vi. 1887-9.

[1] A very complete historical review of the subject is to be found in a paper on the 'Etiology of Croupous Pneumonia,' by Dr. Sternberg, 'Lancet,' 1889, i. p. 370 *et seq.*, to which we are much indebted, and shall have occasion to refer hereafter.

[2] Klebs, 'Arch. exp. Path.' 1877; Eberth, 'Deutsch. Arch. klin. Med.' 1881; Koch, 'Mitth. Gesundheitsamt,' i. 1881.

[3] Virchow's 'Archiv,' lxxxvii.

found to be invariably present, sometimes in great abundance, in the lung of cases of typical lobar pneumonia. He had examined in all eight such cases consecutively, and never missed finding this microbe, which he thought was identical with that previously described by Eberth. It was of an ellipsoid shape, about one micromilleter in length and one-third that in breadth. It stained well with aniline ; it did not occur in colonies, but in pairs, and chains in which the pairs could be discernible. It was found in the midst of the fibrinous exudation in the alveoli, more abundantly in the stage of red or red-grey hepatisation than in that of grey hepatisation. He also detected it in one case in the lymphatics of the interalveolar tissue, and, generally, also in the pleuritic exudation. This was, however, but the forerunner to observations which he continued to make, and which he published two years later, with more detail and with especial reference to the effects of inoculation upon animals of the organism which came now to be known by the name of the 'Pneumococcus.'[1]

The observations were made on as many as fifty cases, and confirmed by many others, when Friedländer's second paper appeared,[2] but there had been also an important addition to the morphology of the bacterium in the discovery of a clear capsule or sheath to the microbe. This discovery was first made by Dr. Gunther in a case where the coccus had been found in the pulmonary exudation withdrawn by an exploring syringe from a living subject.[3] A similar observation of the presence of micro-organisms in the inflamed lung during life was also made by Professor Leyden; but in his case the capsule was not demonstrated.

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[1] Although the progress of inquiry has, as will be seen, materially diminished the importance of the rôle originally assigned to this particular micro-organism, yet this work of the late Dr. Friedländer must always hold a prominent place in the history of the subject. The accuracy of his observations has never been disputed, although his inferences have not proved entirely correct.

[2] Gunther and Leyden, 'Abhandl. Verein innere Med.' 1882.

[3] *Ibid.*

The failure in some cases to detect this capsule was explained by Friedländer (who now came to recognise the presence of the capsule as a characteristic feature of 'pneumococcus') as being due to the methods of preparation. It was found that whereas the coccus itself[1] stained deeply with Gram's method (gentian-violet in aniline water, and subsequent immersion of the sections in a weak solution of iodine) the capsule remained quite unstained and transparent. The organism itself assumes more than one form; it may be spherical, or ovoidal, often in pairs (diplococcus) or small chains or even rods, but in each case the clear capsule surrounds the coccus, chain or rod, like a halo.[2] The capsule seems to disappear under certain circumstances, such as an advanced stage of the disease.

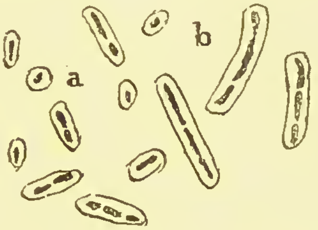

FIG. 14.—*Bacillus Pneumoniæ* (Friedländer). *a.* Oval cells and rows of cells with gelatinous capsules. *b.* Rods with gelatinous capsules (after Ziegler). (× 500.)

The characters of any micro-organism do not, as Koch has so well shown, rest solely upon their size and shape. Such minute organisms may present comparatively little morphological differences and yet belong to totally different classes. The methods of cultivation in specially prepared media (as serum, peptone-gelatine, agar-agar, &c.) serve to bring out striking variations in their mode of growth and in their action upon the media in which they grow. Accordingly, the next step to be taken in determining the specificity of any microbe is to cultivate it. The result of this in the case of Friedländer's coccus was to produce in the tube-cultures in gelatine a characteristic form of growth, in which the track of the inoculating needle was marked by a delicate cloud terminating on the free surface in a rounded prominence, so that the whole culture resembled a round-headed nail. This 'nail-culture' was considered to be distinctive, and it could only be produced by the capsulated coccus. In plate-cultures this microbe, which grows on gelatine at comparatively low temperatures (16° to 20° C.) forms colonies in the form of a thick, white, glistening layer marked by irregularities of the surface, and it does not liquefy the gelatine.

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[1] This is Friedländer's statement, but it appears from later statements by others that this microbe cannot be permanently stained by Gram's method, and that it thus differs from the diplococcus discovered by Fraenkel (*vide* Ziegler, 'Path. Anat.' vol. i. 6th ed. 1889, p. 452, and others).

[2] Other micro-organisms have been shown to possess similar capsules. Klebs thinks the character sufficient to justify their position as a distinct class—Glœococcus (see 'Allg. Path.' p. 328).

In order, however, that Friedländer's discovery should really take the position claimed for it—namely, that the 'pneumococcus' is not a mere concomitant of pneumonia, but that it is causally related to the disease—that, in fact, it should stand in the same relation to pneumonia as the bacillus tuberculosis does to tubercle, it is essential that it should fulfil the conditions formulated by Koch with regard to all pathogenic organisms. Thus, it is not enough that it should be found in close and invariable association with the disease, but, further, it should be capable, when isolated and inoculated in animals, of reproducing the disease in them. The first of these conditions seemed assured by the large number of instances in which Friedländer found this organism in pneumonic lungs. The second condition was fulfilled to his satisfaction by the results of his inoculation experiments. At any rate, he found that when the cultures were injected into the pleura or lung of guinea-pigs or mice, the former frequently, the latter invariably, succumbed with the production of pulmonary engorgement and infiltration, and also with an abundant development of 'pneumococci' in the tissues of the inoculated animal. Somewhat similar effects followed inhalation experiments. It was remarkable, however, that the rabbit appeared to be quite insusceptible to the action of the microbe.

A singular confirmation was given to the statement of the pathogenic character of this micro-organism in the discovery of the 'pneumococcus' in connection with the extensive prevalence of pneumonia in a prison at Amberg by Emmerich.[1] In 1880 Kerchensteiner had reported an epidemic of pneumonia in this prison, which lasted from January to June, and in which 161 prisoners were attacked, 46 dying of the disease. Emmerich, assisted by Frobenius, succeeded in demonstrating in the dust accumulated beneath the flooring of the dormitory in which these cases had occurred, abundance of the pneumococci, and obtained the same results as Friedländer from inoculation of them into animals. The observation was controlled by an examination of other floors in places which had been free from pneumonia, without detecting in them any of these organisms. Moreover, no further cases of pneumonia occurred in this prison after the place had been thoroughly disinfected and cleansed. Cornil and Babes, in relating this experience, consider that it requires confirmation. It is, however, certain that it was mostly regarded as affording considerable support to Friedländer's contention.

It may be here remarked that as the characters of this organism became known to bacteriologists, its position among the class of micrococci came to be regarded as erroneous, for we find it placed by some among the Bacteria,[2] and by others among the Bacilli.[1] At any rate, the fact that it assumes several forms, as rounded or ellipsoidal cocci, diplococci, and rods, in each of which it is invested by a gelatinous capsule, has determined a general recognition of its affinities, being rather with these groups than with the micrococci. And when other more typical cocci came to be described in connection with pneumonia, it seems to have been felt that the term 'pneumococcus' ought no longer to be applied to Friedländer's organism.[2] It is, therefore, now generally spoken of as the *Bacillus pneumoniæ*, or 'Friedländer's bacillus.' Moreover it was not destined to hold its position long as the sole pneumonic microbe. This was, indeed, recognised by Friedländer himself, for in noticing some (then) recent researches of other observers, he remarks on the probability of there being other microbes associated with different types of the disease.[3] The characters of the 'pneumococcus' had hardly been fully described before it was shown to be very frequently absent from the pneumonic lung; whilst, on the other hand, it was detected in the normal saliva and nasal mucus, and in the sputa of other diseases than pneumonia. It was further shown that neither the 'capsule' nor the 'nail-headed' culture was peculiar to it.

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[1] 'Fortschritte der Medicin,' 1884, p. 153.

[2] See Crookshank, *loc. cit.* p. 226.

At a debate upon pneumonia, opened by Professor Jürgensen at the Congress of Internal Medicine at Berlin in 1884, much attention was naturally paid to the comparatively recent confirmation given to the infective theory of pneumonia by the researches of Friedländer and Frobenius. The former contributed to the debate some fresh facts relative to the bacteriology of the subject. At the same time Dr. A. Fraenkel announced that in several cases that he had examined [1] he had isolated an organism which, although bearing a superficial resemblance to that described by Friedländer, differed from the latter in so many respects as to compel the belief that the two microbes were of different nature. This organism was found by Fraenkel in the sputa of every case of pneumonia that he examined. It was a micrococcus, ovoidal or lancet-shaped, occurring mostly in pairs, or in short chains of paired cocci. It was, therefore, named the *Diplococcus pneumoniæ*.

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[1] See Ziegler, *loc. cit.* p. 452.

[2] An analogous change in the original nomenclature of a pathogenic organism is afforded by the microbe considered by Koch to be the cause of Asiatic cholera. He first described this as a 'comma-shaped' *bacillus*, but it came to be subsequently shown to belong rather to the class *Spirillum*. Such changes in nomenclature, although apt to be confusing, are almost inevitable in the study of these very minute organisms.

[3] 'Fortschritte der Medicin,' 1884, p. 654.

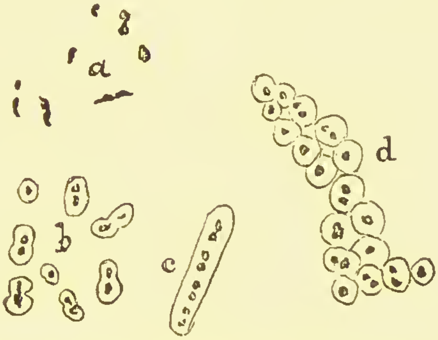

FIG. 15.—*Diplococcus pneumoniæ* (Weichselbaum. Fraenkel). *a.* Cocci without capsules. *b.* Micrococci and diplococci with gelatinous capsules. *c.* Chains of capsulated cocci. *d.* Colony of cocci (after Ziegler). (× 500.)

It, too, was furnished with a gelatinous transparent sheath or capsule, but it could be well stained by Gram's method.[2] In form, then, it differed from Friedländer's bacillus. It differed also in the type of its cultures, in the reaction of the media in which it grew best, and the temperature at which it could be best cultivated.[3] Nor were the effects of its inoculation upon animals the same as those observed in the case of the bacillus. Thus it was found to be capable of producing marked results in rabbits, which, as we have seen, are refractory to inoculations with Friedländer's bacillus. Subcutaneous or intrapleural injection of cultures in these animals caused their death within one to two days, apparently from septicæmia, of which the notable features were a marked swelling of the spleen, and the abundant appearance of diplococci in the blood. When the injection was into the pleura, pleurisy, pericarditis, and sometimes pneumonic infiltration occurred, in the fibrinous exudation of which the diplococci were to be found. Similar effects followed like experiments with mice, but less constantly with guinea-pigs. They showed, however, a marked distinction from the reactions of Friedländer's bacillus, hardly to be explained on the ground of different degrees of virulence of the same organism.

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[1] 'Verhandl. des Congresses für innere Medicin,' 1884, p. 17.

[2] See note, *ante*, p. 341.

[3] Thus it formed in blood serum a gelatinous greyish white layer. In gelatine it required a temperature of 27°, and a faintly alkaline reaction of the medium. Its growth was very easily arrested by alterations in temperature or in this alkalinity. Morever, unlike the 'bacillus,' it was not possible to make potato cultivations.

Meanwhile a very similar series of observations was being made by Talamon[1] in Paris, upon his discovery of a microbe, which has since been identified with Fraenkel's diplococcus. Talamon found in pneumonic lungs, both after death in the stage of grey hepatisation, and by puncture of the inflamed lung during life, a micrococcus of ovoidal shape, or lanceolated, but without any capsule. In two instances only did he detect it in the blood. He found that this microbe was especially pathogenic to rabbits, more so than to guinea-pigs or dogs, its injection into the lung or pleura of the first-named exciting fibrinous pleurisy, pericarditis, and lobar pneumonia, these experiments being, according to Sternberg, the first to demonstrate that pneumonia can be excited by the direct introduction of a microbe into the lung.[2]

Fraenkel soon came to recognise that his diplococcus was not derived in any way from the 'pneumococcus' of Friedländer, and his investigations led him to wider conceptions of its sphere of operation. Thus he found it in the fluid of empyema following pneumonia, and in meningitis. But what was even more remarkable, he proved its presence in normal saliva, not invariably, but sufficiently often to show that its presence there could not be considered as accidental. Bearing in mind the septicæmic effects following its inoculation in rabbits, the analogy with the 'sputum septicæmia' described by Pasteur and Sternberg seemed to be strongly suggested, and although we are not certain that Fraenkel went so far as to identify this microbe with that discovered by Pasteur in the saliva and to which the latter attributed that form of septicæmia, yet its close relationship is probable. Indeed, Sternberg contends that the 'Micrococcus Pasteuri' is the same as the diplococcus pneumoniæ. We shall return to this interesting point later, but at present it may suffice to note that many other observers have confirmed Fraenkel's observation that the diplococcus is to be found in apparently healthy saliva—a fact which considerably diminishes the diagnostic importance of its discovery in sputa.

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[1] 'Progrès médical,' November 1883.

[2] Sternberg, 'Lancet,' 1889, vol. i.

It was not long before the fact appeared that the most frequent of all organisms in acute pneumonia, whether in the sputum or in the inflamed lung itself, was undoubtedly the diplococcus.[1] Perhaps the most important of these researches was that of Weichselbaum of Vienna,[2] which embraced a larger number of cases than any previously. He examined no fewer than 129 cases of inflammation of the lungs, of which 102 were primary and 27 secondary, the former containing 94 examples of croupous lobar pneumonia. Fraenkel's *diplococcus* was met with in 94 cases (80 primary, 14 secondary), Friedländer's *bacillus* occurred in 9 cases (8 primary), and the remainder were associated with the *streptococcus pyogenes* in 21 (13 primary), or the *staphylococcus pyogenes aureus* or *albus* in 5 (all secondary). His description of the characters and reactions of the diplococcus tallies with that of other observers in every respect. But the fact that in some cases of the disease the exciting agent seemed to be another kind of microbe led him to the conclusion that neither the diplococcus nor the bacillus could be regarded as the sole agent in the production of the disease. His demonstration of the diplococcus in one half of his cases of *secondary* pneumonia was also a fact of some importance.

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[1] Salvioli, Foa, Bordoni-Uffreduzzi, and others confirmed this.
[2] 'Wiener Med. Jahrb.' 1886.

Much later Monti[1] found the diplococcus in 15 out of 19 positive results of exploration of lung among 27 cases of pleuro-pneumonia; in 3 others it was found in association with the staphylococcus, and, in one case, with the streptococcus pyogenes. In view of such researches it seems impossible to reconcile the statements of Friedländer with those of other writers without assuming that the fact of both organisms possessing a capsule had led to a confusion between them in the earlier researches. In other words, that the diplococcus has been often mistaken for the bacillus.[2]

In the light of these and similar observations the question arises whether the detection of the diplococcus in the sputum can be considered as having a diagnostic value. Some of the earliest statements upon the presence of micrococci in pneumonia were made from the study of the sputum.[1] Friedländer, on the other hand, examined the exudation in the lung itself, and did not observe the expectoration; whilst, as has been seen, Fraenkel and his followers always succeeded in isolating the diplococcus from sputum. The fact that this organism also exists in normal conditions in the saliva, and that it may be seen in large numbers often in the later stages of a pneumonic attack and during convalescence, although never in such remarkable abundance as in rusty sputa, would seem to deprive its detection of much value in diagnosis.[2] Dr. Klein [3] points out that such sputa contain various septic organisms (streptococci), and that these increase in amount if the sputum be kept for any time. Moreover, so frequent is the presence of the diplococcus in normal saliva or sputa (Gamaleia) that even its predominance is no criterion that pneumonia is present. It is, however, conceivable that in a case of deep-seated pneumonia, where the physical signs are obscure, the occurrence of abundant diplococci would clinch the diagnosis.

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[1] See abstract in 'Centralblatt fur Bakteriologie,' 1888, iv. p. 435.

[2] Dr. Sternberg says (*loc. cit.*), 'Fraenkel's first paper relating to the presence of this micro-organism in pneumonic exudation was published in 1885.' Having ascertained that his own saliva contained this oval micrococcus, he was induced to make an extended and interesting series of experiments, which led him to the conclusion that this micro-organism is far more constantly present in the exudate of fibrinous pneumonia than is the so-called pneumococcus of Friedländer. He says, 'Finally, as regards the relative frequency of the two hitherto investigated micro-organisms in cases of pneumonia, no positive statement can yet be made. Nevertheless, I am inclined to regard the lancet-shaped pneumonic coccus, which is identical with the microbe of sputum septicæmia, as the more frequent and the usual infectious agent of pneumonia, on the ground that this organism is so much more frequently found in the sputum of pneumonic patients than in that of healthy individuals. This conclusion is further supported by the fact that it has not hitherto been possible to isolate directly from the rusty sputum Friedländer's bacillus.'

The most recent and one of the most extended researches on this subject is that of Gamaleia,[4] undertaken in the Pasteur Institute. He identified the diplococcus of Fraenkel and Talamon as the effective agent, but prefers the name *Strepto-*

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[1] Dr. Giles, 'Brit. Med. Journ.' 1883, and 'Collective Investigation Record,' vol. ii. p. 106.

[2] Hunter Mackenzie, 'A Practical Treatise on the Sputum,' p. 15.

[3] 'Centralbl. f. Med.' 1884, p. 529. Klein ('Micro-organisms and Disease,' third edition, p. 74) says that the specific septicæmic micrococcus is not always present in the sputum and lungs of human croupous pneumonia. 'It seems therefore clear that when sputum produces on inoculation disease and death of rodents, this is due to the accidental admixture of a capsulated micrococcus, which, according to Dr. Sternberg of Baltimore, is probably identical with the one occasionally present in the fluid of the mouth even of healthy persons.'

[4] 'Annales Institut Pasteur,' 1888.

coccus lanceolatus Pasteuri for this organism, thus indicating his belief in its identity with the microbe named by Sternberg the *Micrococcus Pasteuri.*[1] Gamaleia made experiments to test its etiological relationship to pneumonia, since it is almost invariably to be found in the lung, blood and sputum of that disease. Thus he found it in 12 fatal cases, and Dr. Goldenberg in 40 successive cases, the identity of the microbe being verified by the results of inoculation of rabbits and mice. The latter were found to be the more susceptible. These researches led to the conclusion that this microbe, while capable of producing septicæmia in the animals named, proves fatal before sufficient time has elapsed for any local affection to be set up. In man, who is less susceptible, there is marked local affection at the site of entrance of the virus (the lung), and less general dissemination. Gamaleia found that in this respect the dog and sheep most resembled man, since injection into the lung of these animals produced a typical croupous pneumonia. He does not think that either Friedländer's bacillus or other organisms have the same relation to pneumonia as this microbe, for he regards the bacillus as a mere saprophyte, and believes that many of the positive results attributed to it are really due to the latter.

There thus appears to be a considerable amount of agreement as to the etiological importance of Fraenkel's diplococcus pneumoniæ, and also as to its identity with the microbe of sputum septicæmia or the micrococcus Pasteuri. Hence it is not surprising that the progress of bacteriological inquiry should have led to the demonstration that its capability of exciting inflammation is by no means limited to the lungs. Literature abounds with records of the detection of this diplococcus in the inflamed products of many serous membranes and internal organs. It is found not only where such inflammations are obviously either concomitant with, or sequential to, acute pneumonia, but also in cases quite independent of that disease. We must confine our remarks on this point within a small compass, but it is essential to take note of these observations before attempting to arrive at any conclusion respecting the true relationship of the diplococcus with the disease we are considering. The affections with which this organism has been found associated are chiefly pleuritis, pericarditis, peritonitis, endocarditis, meningitis, nephritis, and otitis.

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[1] See Dr. Sternberg's paper, 'Lancet,' i. 1889, for a very interesting summary of Gamaleia's research.

Pleurisy.—It need hardly be said that the diplococcus, when found in pneumonic lung, is also to be detected in the pleuritic exudation that usually accompanies pneumonia, for practically in such case the serous inflammation is part and parcel of the pulmonary. It is, however, interesting to learn that, as has been reported by Fraenkel (6 cases), Weichselbaum, Netter, and others, in cases of empyema following on pneumonia, this microbe has been obtained from the pus, and not, as is usually the case, the more common streptococcus pyogenes. It is still more interesting that this particular micro-organism has been detected and isolated in the exudation of primary pleurisy independently of pneumonia, concurrent or antecedent, as it has been both by Netter and Weichselbaum.[1]

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[1] Netter, 'Bull. et Mem. Soc. Méd. des Hôp.' 1889, No. 1. In an exhaustive paper on empyema he distinguishes between the 'metapneumonic' and primary forms. He shows that the first named, which run a mild course, are invariably associated with the diplococcus pneumoniæ, and remarks that this microbe, which is not pyogenic in the lung, is readily pyogenic in serous membranes. He refers all differences which metapneumonic pleurisy may exhibit from other forms of pleurisy, to the fact that it depends on the pneumococcus. At the same time he shows that occasionally there may exist a 'primary pneumococcic pleurisy,' of which he had met with ten examples, in two coinciding with pericarditis, one of these complicating old nephritis. All the rest were simple primary pleurisies. He infers from his own cases that the greater number of purulent pleurisies in childhood are 'pleurisies à pneumocoques.'

Pericarditis.—Almost the same may be said of pericarditis as of pleurisy in this respect, although it has been perhaps less studied bacteriologically. Netter found the pneumonic organism in purulent pericarditis in 1886, and more recently its presence has been described in fibrinous pericarditis complicating pneumonia by Banti,[1] and Thue.[2] We are not aware whether there are any cases of this affection, apart from pneumonia, in which the diplococcus has been met with.

Endocarditis.—The connection of ulcerative or malignant endocarditis with antecedent pneumonia has been known for some time (see *ante*, Chapter VI, p. 108). It has also been long demonstrated that, in that form of endocarditis at least, micro-organisms abound in the valvular vegetations, and in the emboli, which are often widely scattered from that source. In course of time, as the varieties of microbes became differentiated and the class Micrococcus was shown to consist of many species, the identification of these endocarditic organisms with the pyogenic forms, especially the staphylococcus aureus and albus, was made. In relation to the organisms found in croupous pneumonia the most complete account is that recently given by Weichselbaum,[3] who, out of twenty-one cases of endocarditis, proved the presence of the diplococcus pneumoniæ in no less than seven, finding in the other cases as many as six different kinds of microbe including, in one instance, a capsulated organism resembling Friedländer's bacillus. The cases in which the diplococcus was found were as follows :—

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[1] Banti ('Deutsch. med. Woch.' 1888, No. 44, abstract in 'Centralbl. f. Bakteriologie,' 1889 v. p. 131) obtained a pure culture of the diplococcus pneumoniæ from a case of pericarditis complicating pneumonia, and in another case where the staphylococcus aureus occurred in the lung as well as the diplococcus, the former organism alone was isolated from the exudation of the pleura and pericardium.

[2] Thue, 'Centralblatt fur Bakteriologie,' 1889 v. p. 31, describes a series of cases where croupous pneumonia was complicated with pericarditis. In four out of five cases the diplococcus was met with in the pleural and pericarditic exudations, as well as in the substance of the serous membranes.

[3] Ziegler and Nauwerck's 'Beiträge zur path. Anat.' bd. iv. hft. 3, 1888. Weichselbaum gives a full history of the subject of micro-organisms in connection with endocarditis before entering on the description of his own researches. Senger (1886) found capsulated cocci in a case of endocarditis ulcerosa complicating pneumonia, but these results are thought to be doubtful by Weichselbaum. Netter wrote ('Arch. Phys.' 1886, and 'Arch. Gén.' 1887) on 'Endocardite pneumonique,' and out of nine cases identified in seven the 'pneumococcus' in the vegetations. He pointed

Case 1.—Endocarditis recurrens ulcerosa of the aortic valves ; old pneumonia ; old infarcts in the right kidney.

Case 15.—Endocarditis recurrens ulcerosa of the mitral valve ; excentric hypertrophy of the left, and dilatation of the right, ventricle ; infarcts in the spleen ; acute cerebrospinal meningitis.

Case 16.—Old endocarditis ulcerosa of the aortic valves ; excentric hypertrophy of the whole heart.

Case 19.—Endocarditis recurrens verrucosa of the mitral valve; double pneumonia ; infarcts in spleen ; parenchymatous nephritis.

Case 24.—Endocarditis recurrens ulcerosa of the aortic valves ; excentric hypertrophy of the left and dilatation of the right ventricle ; infarcts in the spleen.

Case 25.—Endocarditis ulcerosa of the aortic valves ; embolism of the right Sylvian artery, softening of the right Island of Reil and right temporal lobe, and partial leptomeningitis ; infarcts in spleen; old left pneumonia.

In one case (6) this microbe occurred in association with the streptococcus pyogenes and a non-culturable bacillus. In the remaining six cases the diplococcus was unmingled with any other form, and in all but one case (16) the experimental test was applied as well as the microscopical.

It will be seen that in some of the above cases there was no pneumonia, either old or recent, and it would appear that here, as with pleurisy (and pericarditis ?), the 'pneumococcus' may set up independent inflammation of a serous membrane.[1]

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out that it was not necessarily, although most frequently, associated with pneumonia. But in the vast majority of the observations recorded of bacteriological research into endocarditic vegetations where the type of micro-organism was ascertained, the microbe has *not* been the diplococcus.

[1] In a paper on Endocarditis from Pneumonia ('Rev. de Méd.' 1888, p. 328) Haushalter points out that in some cases of endocarditis secondary to an infective disease, the process may be so slow as to make the relationship between the two affections very obscure. He relates a case fatal from

Peritonitis.—It is to Weichselbaum again that we owe information upon the rare dependence of peritonitis, as of primary pleuritis, upon the diplococcus pneumoniæ. He has recorded[1] two cases of primary peritonitis, each of which was associated with pleurisy, in which this microbe was found and proved by cultivation and experiment. The diplococcus occurred in amount proportionate to the intensity of the inflammation, and he sees no difficulty in accepting the etiological relationship. It is to be noted that in each case there was ulceration of the stomach (one cancerous, the other a simple ulcer); but these conditions, although contributing to the peritonitis, were not considered to have excited it *per se.*

Meningitis.—Since the discovery of the pneumococcus by Fraenkel a large share of attention has been given to the occurrence of this organism in the exudation of meningitis both when complicating pneumonia and also when apart from that disease. The occurrence of meningitis in pneumonia is undoubted, and supported by many recorded cases, and it may be remarked that Klebs, who first found micro-organisms in pneumonia, met with them in the meninges and ventricular fluid of the same case. One of the earliest of Fraenkel's papers[2] contained a description of a case of cerebro-spinal meningitis complicating pneumonia in which the diplococcus could be cultivated from the meningitic exudation. Weichselbaum, in referring to this paper, says that he himself had observed two cases,[3] and

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abscess of the lung following pneumonia, where a small focus of cocci was found on the mitral valve, which, had it not been for the death of the patient, might conceivably have initiated a progressive endocarditis having its origin in the pneumonic attack.

[1] 'Centralbl. f. Bakter.' v. 33, 1888.

[2] 'Zeitsch. klin. Med.' xi. and 'Deutsch. med. Woch.' 1885, No. 13.

[3] 'Centralbl. f. Bakter.' i. 79, 1887. The same writer ('Fortsch. d. Med.' 1887, Nos. 18 and 19) reviews the whole subject of the etiology of cerebro-spinal meningitis, and describes not only the diplococcus pneumoniæ in this connection but also another kind of bacterium, which he terms 'diplococcus intracellularis meningitidis.'

similar experience has been gained by Foa, Bordoni-Ufreduzzi,[1] Senger, and others. In a later communication Foa and Bordoni-Ufreduzzi relate their experience of an epidemic of cerebro-spinal meningitis in 1886, in which some cases were associated with pneumonia and others not. They suggest that it would be preferable to denote the microbe common to these and other diseases by a term more neutral than 'pneumococcus' or 'meningococcus,' such as diplococcus lanceolatus s. capsulatus, to which the affix 'pneumoniæ,' &c., might be applied in reference to the disease in which it is present.[2]

Netter[3] has shown that several different kinds of microbe may be met with in suppurative meningitis, of which he distinguishes the two classes of 'direct' and 'metastatic,' the former being produced by extension from the naso-pharynx or the auditory passages, the latter arising in the course of infective diseases. In no fewer than eighteen cases out of twenty-five did he find this microbe to be the diplococcus pneumoniæ (or, excluding six in which pneumonia was also present, in ten out of nineteen). And out of forty-five cases of meningitis recorded in literature he finds that the diplococcus is stated to have been present in twenty-seven, a striking confirmation of his own results. Since we are not dealing here with the pathogeny of meningitis apart from pneumonia, it is not necessary to mention the other organisms that Netter and others have met with in this disease, beyond the fact that Weichselbaum's discovery above alluded to is confirmed both by Netter and Goldschmidt.[4]

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[1] 'Deutsch. med. Woch.' 1886. They found a coccus in four cases of meningitis (two complicated with pneumonia), identical with the diplococcus pneumoniæ.

[2] See abstract in 'Centralbl. f. Bakter.' 1888, iv. p. 40. Subsequently Foa appears to have modified this view, since he declares that there is a difference between the two organisms (*ibid.* 1889, v. p. 384).

[3] 'La France Médicale,' 1889, No. 84. His previous paper is to be found in the 'Arch. Gén. de Méd.' 1887.

[4] 'Centralbl. f. Bakter.' 1888, ii. 649.

It is noteworthy that Fraenkel early pointed out the existence of his diplococcus in saliva and nasal mucus, so that it is not improbable that many cases of meningitis, apparently due to the diplococcus pneumoniæ, may arise by extension from the nasal cavities and sinuses connected therewith. The same explanation may also be applied to such cases as are secondary to disease of the ear, since the diplococcus has been found in otitis, to which we may now briefly refer. At the same time it is possible that in cases of meningitis secondary to pneumonia the infection of the meninges takes place, as in other organs, through the medium of the blood.

Otitis.—The chief contributions to the question of bacterial infection in inflammatory affections of the middle ear are those of Zaufal,[1] who first discovered the diplococcus pneumoniæ in a case of otitis media following typhoid. He has since met with similar association; but of course other pyogenic organisms sometimes occur, including Friedländer's bacillus. Scheibe,[2] in eleven cases of middle-ear disease, found as many as seven different kinds of micro-organisms, including the diplococcus pneumoniæ.

Nephritis.—The comparative frequency with which albuminuria is met with in the course of acute pneumonia lends additional interest to the records of Senger and Klebs of nephritis associated with micro-organisms in cases of pneumonia;[3] but there is not much confirmation of these observations. Cornil and Babes[4] content themselves with the statement that infective nephrites are fairly common in pneumonia, and refer to the discovery by Bozzolo[5] of the capsulated diplococci of pneumonia in nephritis (white kidney) accompanying pleurisy and peritonitis.

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[1] See abstract in 'Centralbl. f. Bakter.' 1889, v. and vi.
[2] *Ibid.* vi. 186.
[3] Klebs, 'Die allg. Path.' I. Jena, 1887, p. 331.
[4] 'Les Bactéries,' 2nd ed. p. 379.
[5] Bozzolo, 'Centralbl. f. klin. Med.' 1885, 11.

We have now passed in review some of the very abundant records respecting the part played, or considered to be played, in disease by Fraenkel's diplococcus. We have seen how widespread is its distribution, and how it can by no means be regarded as special to pneumonia alone. Nor, on the other hand, is there any agreement as to this particular microorganism being the sole microbe found in the inflamed lung itself. It is one—perhaps the chief—agent responsible for exciting the pulmonary inflammation, just as in many other inflammations (especially of serous membranes) this diplococcus seems to predominate. It has been clearly identified in the saliva of healthy subjects, and shown to excite on inoculation in some animals septicæmia, in others an affection more comparable to the pneumonia of the human subject. Thus it is manifest that, whatever be the precise etiological relationship between the diplococcus and pneumonia, it cannot be of the same kind as that believed to subsist in the case of some other so-called specific organisms and the diseases with which they are associated (*e.g.* tubercle).

The subject unquestionably requires further elucidation, since not only are statements upon this relationship conflicting, but it has been shown by Weichselbaum that the *rôle* of the diplococcus is not confined to cases of primary croupous pneumonia, but that it is to be found in association with several forms of secondary pneumonia. A recent writer[1] has indeed detected both Friedländer's bacillus and Fraenkel's coccus in acute *broncho-pneumonia*, and upon this founds a claim for regarding the conditions of lobar and lobular pneumonia, though anatomically distinct, as essentially one and the same disease. Yet, in view of all the facts of etiology and clinical history of these two affections, it is difficult to admit such unity. On the other hand, according to the results of bacteriological inquiry, it would appear that the condition of primary lobar pneumonia can not be regarded as invariably due to a single virus, and that cases anatomically as well as clinically indistinguishable arise in various ways.

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[1] Massolongo, 'Gazetta degli Ospedali,' 1887, No. 86, abstract in 'Centralbl. f. Bakter.' 1888, iv. p. 624.

But the matter does not end here. We have seen that Weichselbaum and others have found certain pyogenic microbes in cases of pneumonia, primary as well as secondary (see *ante*, p. 347), and it is not disputed that in some instances the organism discovered by Friedländer may alone be present, although the majority of cases exhibit Fraenkel's diplococcus. There must now be added to the list another species of bacterium which Dr. Klein has discovered in the sputa and lungs of cases occurring in the severe epidemic of pneumonia at Middlesbrough in the spring of 1888 (see Chap. VI, p. 267), and which has formed the subject of an inquiry by Dr. Ballard on behalf of the Local Government Board.[1] Clinically and anatomically the cases do not appear to have differed in any important respect from the usual type of lobar pneumonia, except it be in the very high mortality. But the bacteriological results differed from those ordinarily met with. Neither Friedländer's bacillus nor the diplococcus pneumoniæ was observed, but an organism consisting of short oval rods, isolated, or in pairs or short chains, and also in the form of longer bacilli. The organism was enclosed in a sheath, and the extremities of its protoplasm stained more deeply than the rest of its substance. It occurred in enormous numbers. Dr. Klein gives full details of its characters on cultivation, and of the effects of its inoculation in animals, where it produced hæmorrhagic pneumonia, pleurisy, pericarditis, and sometimes peritonitis. He obtained a remarkable confirmation of its virulence and pathogenic property in the outbreak at his laboratory of a severe and fatal epidemic of pneumonia among mice, guinea-pigs, and monkeys, which was proved to have been due to this bacterium. In every instance there was well marked hepatisation of the lungs.[1]

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[1] Report of medical officer of the Local Government Board, 1888-9. For Dr. Klein's report see also 'Centralbl. f. Bakter.' 1889, v. p. 625.

In conclusion, we are of opinion that, so far as regards the bacteriology of pneumonia, there is as yet hardly sufficient evidence to establish the dependence of the disease upon one specific virus. The arguments advanced by Dr. Sternberg in support of the view that the microbe most responsible for exciting the actual inflammation of the lung is a normal denizen of the buccal secretions are of great weight. His view would harmonise well with the doctrine that before infection can occur there must be a condition of lowered vitality on the part of the individual rendering him prone to be affected by the virus, or, at least, unable to resist its action. But this is hardly the same thing as regarding any particular microbe as the sole specific agent of the disease. Nevertheless, it must be considered as proved beyond dispute that inflammation of the lung, like inflammation in general, is associated with the growth and development of certain micro-organisms, which are more virulent in some animals than in others. It has also been proved that these organisms may infect organs and tissues, since they may be found in the inflammatory exudations and products of these parts secondary to an attack of pneumonia ; whilst the fact that these same microbes occur in inflamed serous membranes and elsewhere, apart from pneumonia, would show that their pathogenic action is not limited to this disease. Even then, however, it would still be possible to consider that their association with pneumonia is accidental rather than causal ; that they are epiphytes on a soil already prepared for their sustenance. We shall revert to this subject immediately in speaking of the Pathology of Pneumonia.

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[1] Mosler, 'Deutsch. med. Woch.' 1889, Nos. 13 and 14 (abstr. in 'Centralbl. f. Bakter.' v. p. 832), has also met with another hitherto undescribed form of bacterium in pneumonia. The case in which the microbe was found was one of a small series in which apparently contagion was the cause of the attacks (for details of these cases see Chap. XVI, p. 333). The bacterium was obtained by puncture of the lung during life, and could not be found in the sputa. It somewhat resembled the organism of rabbit septicæmia. But inoculation experiments were negative.

CHAPTER XVIII

PATHOLOGY

Pneumonia as an inflammation—Its distinctive characters—Not a local inflammation merely—Its origin often independent of exposure—Comparison with herpes, quinsy, erysipelas—Specific character—Agency of microbes—Place of pneumonia among acute diseases.

In the present chapter we propose to gather up and put together the substance of much that has gone before. If the account already given of the causes, forms, and associations of pneumonia is, on the whole, faithful and adequate, the rest might safely be left to the reader. Yet, in deference to the fact that the pathology of this disease is still open to controversy, we may here set down the conclusions to which we have ourselves been led.

Whatever opinion be entertained of the proper scope and limits of pneumonia, it will at least be admitted that in the typical form of the disease the phenomena of inflammation are conspicuously exhibited. Both the clinical and the anatomical requirements are satisfied. There is pyrexia, there is exudation with the migration of leucocytes, and there is excess of tissue change by proliferation. Not only so, but the fibrinous exudations which attend pneumonia, the pleurisy which is constant, and the pericarditis which is not rare, bear similar testimony, which is still further corroborated by the character of the allied diseases. Not only is pneumonia an inflammation, it is the pattern and model of inflammations, and, in the history of medicine, has often been referred to in that character.

Moreover, considering the structure of the organs concerned, the function they serve towards the blood, and the effect upon the circulation, even from a mechanical point of view, of such an occurrence as hepatisation, it could never be supposed that the symptoms of pneumonia would be limited to the lung. Not only must such a disease have general as well as local signs, but the precise manner of it, its abrupt origin, uniform duration, and sudden departure, might find explanation also in the sudden invasion, limited term, and rapid resolution of its inflammation products, events which we actually observe with more or less precision.

It is to be noted, at the same time, that pneumonia regarded as an inflammation, is not a process that can be induced by artificial means (p. 326). Neither by wounds nor irritant inhalations, nor in any other way, can the disease be set up, or anything at all resembling it. It must come. Nor have we here, as in the case of traumatic inflammations, a local change first, and next, as in response to this, a set of phenomena which are to be regarded as its proper consequence. On the contrary, the pyrexia will sometimes, as we have seen, precede, and by several days, the earliest physical signs of change within the lung itself.

Nor, in their further progress, is there any precise correspondence between the pyrexia and the local affection. The one is sudden in its origin, regular, or at least conforming more or less to a rule in its course, and abrupt in its departure; the other advances by degrees, unequally in different parts, and, having attained a certain point, is gradually removed by the process of resolution. In no certain or constant relation to the first access of pyrexia, an exudation takes place which, becoming more and more fibrinous, soon solidifies and becomes moulded into its place. Often the area of lung at first affected widens as the disease goes on, and sometimes from a small patch of lung the process spreads so as to involve nearly the whole of it. Whether this is so or not, it is seldom that the part affected, be it large or small, is affected throughout simultaneously. While one portion is œdematous, another is sealed up with solid material, and another beginning to discharge its alveoli of their now liquefied content. True, the whole portion concerned is implicated similarly, but the different parts exhibit different stages of one process. There is thus no moment when the local disease passes as a whole from hyperæmia to consolidation, or as a whole from red hepatisation to grey infiltration.

The same may be said of the process of restitution. It may remain incomplete when the health of the patient is pretty well restored. The lung clears up by degrees, and bit by bit ; the fever leaves of a sudden, sometimes before, sometimes after, and sometimes concurrently with resolution.

Now, from this coincidence of several stages or degrees of solidity in the same lung, we should be led to expect, on the hypothesis of a local inflammation, not a definite course of pyrexia, but a series of elevations and depressions succeeding each other irregularly as fresh portions of lung became implicated or as the local changes proceeded faster at one spot and slower at another. And not only would the pattern of any temperature-chart indicative of the natural course of lung inflammation be continually disfigured by this concurrence of its several stages, but sometimes its earlier phenomena ought to recur. In double pneumonia, for instance, so soon as the second lung began to suffer, as its fellow had suffered before, we might expect, but for the teaching of experience, a second rigor, together with a repetition of the early temperature. We see no such thing. The local inflammation, in its gradual extension and composite characters, offers no sort of parallelism to the fever which for a while accompanies it.

Add to this that the area of lung involved in the pneumonic process is no reliable measure of the severity of the disease. A certain degree of correspondence between the local and the general signs is, indeed, a necessity, for where a considerable portion of the respiratory apparatus is rendered useless the breathing must suffer accordingly. But there is no closer agreement. A small locality of lung inflammation may coincide with a severe and even fatal attack, while a larger one may be free from any serious symptom.

Considerations such as these are opposed to the view that pneumonia is nothing more than a local inflammation; they lend support to the belief that it is to be regarded rather as a specific disease having its chief seat in the lung. Yet to this latter opinion Dr. Wilson Fox objected upon consideration of the manifold causes of pneumonia. 'The disease,' he says, 'may originate under such conditions that it seems impossible to attribute it to any single blood poison.'[1] And truly, in the wide sense in which the term pneumonia is applied by this author, the conditions are sufficiently diverse, and this very diversity, as we have endeavoured to show, tells in favour of a stricter limitation of the word than Dr. Fox would have admitted. The inflammation products in connection with tubercle, with hypostatic consolidation, with alveolar catarrh, with collapse, all these, histologically regarded, may be indistinguishable from pneumonia; but their life history is quite different. On the other hand pneumonia, in the sense in which we speak of it, is entitled to separate and distinct recognition. As with quinsy, with herpes, with facial erysipelas, with acute rheumatism—affections with which it may be justly compared—its modes of origin, subjects, and associated diseases, are known and constant.

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[1] Reynolds' 'Syst. of Med.' vol. iii. p. 675.

Take for example the case of quinsy, and place it side by side with pneumonia. In the character and duration of pyrexia, the rapid recovery or 'defervescence,' the severity of the general as compared with the local symptoms, and the age of the patients, the two affections are strikingly similar. Aching limbs, prostration, chilliness, and often distinct rigor, accompany or sometimes precede a quinsy where the local inflammation is insignificant. And not only is the pyrexia well marked and sometimes extreme, but the course of it, in respect of an early rise to its highest point and rapid 'critical' decline about the fifth or sixth day, resembles nearly the temperature tracing of pneumonic fever.[1] The diminution of chlorides in the urine and increased urea discharge may be mentioned as further points of likeness.

Now, just as pneumonia differs from some other forms of pulmonary consolidation, so does true quinsy differ from other forms of tonsillitis. It is a familiar experience to observe one or both tonsils undergo a gradual increase in size, and at length even suppurate, while no active disturbance accompanies the change, nor, indeed, any symptoms whatever except those that arise immediately from mechanical causes.

Not less pertinent is the case of erysipelas in its traumatic and non-traumatic forms. Traumatic erysipelas, due to a poisoned wound, gives rise to a pyrexia of its own, spreads indefinitely, and is quite uncertain as to its duration, course, and end; but the facial erysipelas known to the physician arises from causes very similar to those that give birth to pneumonia, from cold, exposure, and bad drainage; it has a definite course, special pryexia, and spontaneous recovery.

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[1] In a girl of seventeen, admitted to the Westminster Hospital with quinsy, temperature rose rapidly to 105·8, from which there was a critical fall of great regularity measured hour by hour, until, in twelve hours, it had descended to 99° (see Chart 4, p. 41). The local symptoms gave no trouble, and the patient was soon convalescent. 'West. Hosp. Reports,' vol. ii. p. 14. Wunderlich, *loc. cit.* p. 386.

Furthermore, it seems probable that the essential phenomena of pneumonia may be fulfilled when the seat of inflammation is the pleura rather than the lung. It is admitted, indeed, that pleuritis and hepatisation are joint factors in almost every instance of unequivocal lobar pneumonia, but it is admitted as well that there is no constant relationship between the two, a given extent of hepatisation concurring sometimes with a thick and extensive layer of pleural lymph, and sometimes with only a thin film of it. And in exceptional instances it would appear, further, not only that hepatisation occurs without any pleurisy at all, but also that pleurisy occurs without any hepatisation, and that in both instances alike the clinical history and morbid associations may point to a general disease pathologically identical with pneumonia.[1]

A further illustration is herpes, and it is, perhaps, the best, inasmuch as there is an undoubted affinity between it and pneumonia. There is the labial herpes of a common cold or of some excess in food or drink, which, except as the outward signal of these events, has no significance at all; and there is the same herpes which, whether with or without pneumonia, is preceded by a definite fever of limited duration, and sudden both in its development and defervescence. When herpes and pneumonia concur in the same subject, little regard is paid to the former, and it is not probable that it exerts any influence of its own. But either of these affections may happen singly with precisely similar phenomena. Herpetic fever and pneumonic fever are indistinguishable; and it happens not seldom in the course of a pyrexia, reminding us of pneumonia, that while we are seeking for some local signs of that affection a vesicular eruption about the lips and nostrils makes its appearance instead, and the lung escapes altogether.[1]

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[1] A woman, aged 40, was brought into the Westminster Hospital March 7, 1890—a cook, well-nourished, temperate, and with good personal history, having a temperature of 104°, rusty sputum, and physical signs taken for lung consolidation, with some effusion. She had been ill only six days, and for the last two delirious, and she died the day after admission.

Post-mortem.—A thick layer of soft lymph was found covering the parietal and visceral layers of the right pleura, and the pleural sac contained about a pint of pus. The lung was carnified, but on incision showed no trace of consolidation; its apex was œdematous. The left lung was friable, but presented no other sign of inflammation. *The pia mater of both brain and cord were in a condition of suppurative meningitis.*

For reference to connection between pneumonia and cerebro-spinal meningitis see pp. 120 and 354.

This parallelism between herpes and pneumonia is further established in the relation of both to tonsillitis. For just as labial herpes is a not infrequent companion of pneumonia or, as it were, a substitute for it, so does a precisely similar vesicular eruption sometimes affect the tonsils and pharynx. The course of this herpetic tonsillitis is similar to that of quinsy or of pneumonia, which latter, indeed, is occasionally present as well.[2]

The interrelation of the diseases referred to, of erysipelas, tonsillitis, herpes, and pneumonia, was carefully observed by the Collective Investigation Committee (p. 33). The inquiry was not free from ambiguity, but the general conclusions may be accepted, and they are these: No disease occurs in conjunction with pneumonia so frequently as pneumonia itself. The coincidence of two or more cases of the disease in the same house was found in the proportion of 1 to 8·3. The only other affections apt to concur with pneumonia in the same house are tonsillitis and bronchitis. But the frequent occurrence of erysipelas *in the same district* with pneumonia is noteworthy. Thus in one series (A) of 350 cases erysipelas was prevalent at the time in 70; in a second (B) of like number, it was prevalent in 55, and in a third (C) in 63. The only affection, it is stated, at all parallel with pneumonia in respect of relative frequency is herpes. But it is obvious that so trifling an ailment as this must frequently escape medical observation. According to the same statistics there is no similar correspondence between any of the infectious fevers and pneumonia, and the rarity of diphtheria is noticed.

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[1] See 'Westminster Hospital Reports,' vol. ii. page 177, 'Five cases of pneumonia without physical signs,' reported by Dr. Syers, medical registrar. 'In each case the onset of illness was sudden, the temperature high and falling by crisis; labial herpes was present, and both aspect and pulse-respiration ratio were typically those of pneumonia.'

[2] In the first edition of this work the analogy between acute pneumonia and other acute diseases was further amplified, and, in particular, the case of acute articular rheumatism was instanced as occurring in like subjects, at the same age, sometimes in conjunction, and (according to the observations of Zimmermann and others) with a similar hyperinotic condition of blood. These illustrations, with some other matters referring to the pathology of acute nephritis, are now omitted, not that they have been discredited, but because the examples here given are more direct and apposite, and sufficiently serve our purpose of showing the kinship of acute pneumonia with a large class of acute diseases.

Yet of pneumonia, as of these other affections with which we are comparing it, it must be freely admitted that when contrasted with the specific fevers the points of difference are not less conspicuous than those of likeness. With a duration more or less definite, pneumonia is not, or at the most is very rarely, contagious; it is seldom in a strict sense epidemic; has no fixed period of incubation and no skin eruption. To lay stress upon certain rare examples having an appearance of contagiousness; to force a comparison between the occasional labial herpes of pneumonia and a true febrile eruption; or to confound the exceptional frequency of the disease due to weather causes with a true epidemic, such expedients indicate only anxiety to justify a preconceived opinion. No unbiassed judgment will be influenced by such arguments.

Still the question presses—Granting that pneumonia is not a purely local affection of the lung, in what precise sense is it to be regarded as a general disease? The point is no new one. More than two centuries ago, as related at the beginning of this book, Huxham employed the term 'pneumonic fever,' and in the last century, as Dr. Loomis and others have pointed out, pneumonia was held to be a specific disease (Loomis, Art: Lobar Pneumonia, Pepper's 'Syst. of Med.,' reference to C. Strackius, Nov. Theo. Morg., 1786).

In our own day the name of Jürgensen is especially associated with this teaching. A writer who did much to give clear expression to the grounds for regarding pneumonia in the light of a general disease was the late Dr. Austin Flint, who read a paper on this subject before the New York Medical Society in 1877. He thus sums up his views in his 'Treatise on Medicine' (6th ed. 1886):—

'Acute lobar pneumonitis in the nosological systems of the present as of past time is placed among the local diseases, and in regard to certain questions it has been regarded as the type of a purely inflammatory affection. This view of its pathological character is now held to be erroneous. The pulmonary affection is doubtless inflammatory; but it is the local manifestation or the anatomical characteristic of an infectious febrile disease, sustaining to the latter a relation analogous to that which the affection of the solitary and agminated intestinal follicles sustains to typhoid fever. If this doctrine be true, the proper place for the disease in nosology is among the essential fevers.'

Dr. Flint therefore preferred the term 'pneumonic fever' to that of acute lobar pneumonia, thus reverting to the nomenclature of two hundred years ago.[1]

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[1] It is to be noted also that this doctrine, which was propounded in the first edition of this work, has received the unqualified support of Dr. T. H. Green, who writes (Quain's 'Dict. of Med.' art. Inflammation of Lungs):—

'Acute pneumonia is undoubtedly to be regarded as a general disease, of which the pulmonary inflammation is the prominent local lesion. The

If, then, pneumonia is to be regarded in this light, it must be *zymotic*, using that term in its widest sense.

The difficulties in the acceptance of this view are etiological as well as clinical. Much turns upon the actual origin of the disease and the circumstances that precede its onset. Of these the most prominent is chill, which, however it be minimised, must be admitted to play an important part in causation as we have shown, although not the sole part. The very diversity of its immediate antecedents suggests that these really occupy a secondary place in causation—that they are predisposing, and not essential causes of the disease. If we were to seek amongst them for a common factor in which they were all concerned, it would be found in a lowering of the vitality, and the lessened resistance of the organism to morbid influence that they induce. This may be true even in respect to chill, momentary and transient as its operation may seem to be, and speedily as it is followed by the symptoms of the disease. If pneumonia be due to a virus, and therefore a distinct and specific disease, then no other conclusion is admissible with regard to the agency of chill, alcoholism, and other assigned causes.

It may be urged that this doctrine receives fresh and independent support from the bacteriological researches detailed in the last chapter. Yet even from this standpoint it would appear as if the disease, although mostly dependent upon one special microbe—the diplococcus—is occasionally found, as we have shown, to be associated with other kinds of micro-organism.

It remains to be seen how far the conception of pneumonia

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view that it is a strictly local affection of the lung, to which the pyrexia and other symptoms are secondary, is altogether untenable.'

On the other hand, even those who still hold to the 'untenable' view have been forced to consider whether there is not some special factor at work to produce a type of inflammation which cannot be evoked traumatically or by irritant inhalations and of which the etiology is so varied.

is affected by these divergent statements. It is of course open to argument that the concurrence of bacteria is not essential but, so to speak, accidental (see p. 360). But the proofs of the causal relationship of the diplococcus (and also, it must be confessed, Friedländer's bacillus) are similar to those which are held to be sufficient in the case of other specific diseases. Since these discoveries were made known observers have never failed to find such microbes in the pneumonic lung, or in the sputa, in the pleural exudates, and in the inflamed tissues of other organs in pneumonia. Inoculation experiments have been successful in exciting the disease in other animals—mice, guinea-pigs, or rabbits. It might almost be said that in pneumonia we have a test example of a germ disease, so closely does it seem to conform to the requirements laid down by Koch as proof of such an origin.

The occasional and undoubted occurrence of epidemics of pneumonia, and the evidence of contagion in certain instances, would lend support to such a view, were it not for the fact that contagion is so rare, and that in one authentic instance at least (that recorded by Mosler, v. p. 333) not the diplococcus, but another variety of microbe, was found in connection with the disease. Dr. Klein's researches in the Middlesbrough epidemic also point to other organisms than the diplococcus being associated with it. It would seem, then, as if we should be compelled to abandon the notion that pneumonia is always of the same nature, and admit that it may be evoked by more than one poison. This would harmonise with the supervention of pneumonia in the course of specific fevers, where it may be attributed not to the diplococcus but to the virus of the diseas which it complicates (p. 225).

Assuming, as the bacteriologist has a strong reason for asserting, that in ordinary primary lobar pneumonia the diplococcus is to be regarded as the causal agent, we are met by the arguments of Dr. Sternberg, already referred to, that this special microbe is often to be found in human saliva, being, in point of fact, none other than the micrococcus Pasteuri. How far, it may be asked, does this admission militate against the notion that pneumonia is a specific affection? At first sight it might seem absolutely fatal to it, for what more likely than the detection in the pneumonic sputum of a microbe, usually, but not invariably, present in normal buccal secretions. But it must be remembered that it is not ordinarily found in the bronchitic sputa nor in that of phthisis, that it is to be detected in the hepatised lung, and, what is more convincing still, in the inflammatory products of other regions, and even in circumstances where pneumonia itself has not been excited (*e.g.* cerebro-spinal meningitis). Can it be that, given a particular conjunction of antecedents, or of what we call predisposing causes, of which chill and exposure are the most prominent, but which, in the case of epidemics, may conceivably be meteorological, that this microbe, already suitably placed for invading the body by the respiratory tract, finds the conditions suitable for its development and excites the inflammatory phenomena in the lung with which we are familiar?

In reviewing the evidence, clinical as well as etiological, in favour of the belief of pneumonia being a specific disease, it is impossible not to admit that there is much in its support. But, in face of the fact that more than one kind of organism has been claimed to be causative of the disease, and of the comparatively recent discovery of such pneumonic microbes, we may be pardoned for withholding an unqualified assent to the proposition that pneumonia is a germ disease.

The whole argument from this point of view may be summed up in a few words :—

(1) Pneumonia is a specific general disease, of which lobar inflammation of the lung is the local manifestation.

(2) The most constant cause of pneumonia is Fraenkel's diplococcus, but there are other viruses which may evoke it.

(3) Chill and other causes, some more some less distinctly observed in that relation, are to be regarded only as secondary or predisposing.

(4) The 'pneumonic virus' may, and often does, excite inflammatory disturbance in other organs than the lung; and sometimes such inflammations may be excited without the lung participating.

(5) Exceptionally, pneumonia may be contagious.

Time will show whether or not these conclusions will be finally accepted. In any case, we may rest assured that the main clinical features of pneumonia will remain in spite of changing theories, varieties of technical expression, and even actual diversities from time to time in the precise form of the thing itself. The face of disease does not change, but it has more than one expression, and we cannot discharge from our descriptions all that is accidental and ephemeral. Yet to us, as to former generations, it is apparent that pneumonia occupies a middle place between specific fevers and local inflammations, and has something in common with both. It falls short of the one by wanting the material of infection and a period of incubation; it exceeds the other by possessing the character of orderly pyrexia, definite duration, and severity in large degree independent of its extent. It is not a fever which, apart from any fixed seat, pervades the system generally, like typhus, and, in default of definite lesion, is laid to the charge of the blood; neither is it a mere pyrexia, the immediate response to a local inflammation. For its full recognition (we do not say for its essential existence) lung inflammation is necessary, but the presence of such inflammation by no means implies or secures the presence of pneumonia. At one time it will present, almost exclusively, the characters of a local disease;

at another, chiefly those of a specific poison. By its aspect, its duration, and concomitant lesions, as well as by the special character it acquires in certain epidemic constitutions, it would seem most like fever; in its actual histology, its obvious origin from chill, near connection with chill diseases, and the absence of any incubation period or of extraneous material cause, it is most like an 'idiopathic' inflammation. And this position of isolation between distinct classes of disease is further secured to pneumonia by the fact that, in the large majority of cases, the lung itself does not participate structurally in the changes that occur within it. In yielding directly from the blood a material destined to be removed from the system without injury to the organ that receives it, the pneumonic process is in fact special and *sui generis*. Considerations derived from its germ origin are of the highest interest and importance; but even when these are exhausted the disease must be judged of by itself, less by comparison and analogy, which may be forced and misleading, than by endeavouring to trace from the pattern of nature its own proper history in various circumstances, careless whether in the delineation we are producing a picture in harmony with preconceived notions.

PART IV

TREATMENT

CHAPTER XIX

HISTORY AND STATISTICS OF VARIOUS MODES OF TREATMENT

The numerical method of estimating the results of treatment—Its difficulties—Rates of mortality at various periods—Lessened mortality of the disease in the present day—Treatment by bleeding—Antimony in large doses—Practice of Laennec and Andral—The 'jugulant' method—British practice early in the century—Schism—'Expectant' treatment—Views of the late Dr. Hughes Bennett—The 'restorative' treatment—Statistics—Asserted action of various drugs.

THE subject of treatment—of the mode of influencing beneficially the natural course of disease—is just now especially difficult and embarrassing. Particular drugs can no longer be recommended on the ground of usage or the authority of great names. Conflicting views as to the virtues of the materia medica are not now to be settled by the use of *à priori* arguments. It is easy to give good reasons for bad remedies. We can trust only the teaching of experience.

Of such teaching—or of teaching that claims to be such—there is, indeed, no want, as we shall presently see, in the literature of pneumonia. Yet, in spite of a mass of evidence of this kind, it is doubtful whether the statistical method can ever be successfully applied to the question. If it were possible, indeed, for the same observer to collect examples numerous enough for the comparison of like cases one against the other; and if, with that material, he could put in practice without favour or prejudice, at one period and in one set of circumstances, several modes of treatment towards as many groups of patients all similarly affected, he might possibly arrive at conclusions sufficient at least for his own guidance. But the comparison of the results of one observer with those of another; of the same disease at different periods and seasons and places; of cases treated upon a plan which is in favour with those treated upon a plan that is distrusted—in a word, comparison and tabulation under the only conditions possible—can never be trustworthy or conclusive.

Even supposing that someone might be found practised enough to be reliable, yet sufficiently unbiassed to deal equally with conflicting methods, the result of his labour, however ample the field, must still be incomplete. At the most no more would be determined than the relative value of some two or three modes of treatment; not the best method would be thus arrived at, but only the comparative merits of a very few methods, no one of them necessarily good.

Again, it is very difficult to estimate by mere narration the comparative severity of cases, so as to set them fairly one against the other. Comparison is hardly possible without personal observation, while it is hardly just unless the cases compared are subjected to the same external influences—happen, that is, at the same time[1] and place. Yet it needs not one but many observers, not a single occasion but a wide stretch of time, before a sufficient body of examples can be collected to form the material for general conclusions.

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[1] The discrepancies between the mortalities at *various periods* under similar treatment and with the same physicians are remarkable. Thus Bamberger during six years had a mortality of 11·2 per cent. for the first three and of 18·19 for the second three. Brandes of Copenhagen found the rate vary from 5·4 to 31 per cent. in two successive years. In 1858 the mortality at St. Thomas's was 17 per cent. and in 1859 it was 5·7 per cent. Other reports might be quoted to like effect. See Dr. Peacock's

For such reasons as these the method by counting, which at first commends itself as the best, is in fact untrustworthy, the necessary conditions being unattainable. Every advocate of a particular remedy finds numbers on his own side. They are more easily invoked to support foregone conclusions than used for the actual discovery of truth.

These considerations, applicable to all diseases for which no specific remedy has been found, are of special cogency in the case of pneumonia, where, owing to the occasional ambiguity of physical signs, it is sometimes impossible to be assured, not merely of its gravity, but even of its existence. When this is beyond dispute there is still the need of an even comparison of cases. Infants and children, adults and old people, have separate death rates, while, age apart, the disease is especially to be dreaded in certain subjects and at certain seasons. What can be the value of a statistical table in which all these things are jumbled together?[1]

Nor if these difficulties could be surmounted can we suppose it possible that any rigid system of treatment formally proposed beforehand can actually be put in practice in all its integrity. Whatever the principle of treatment, it must be

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Report on Cases of Pneumonia, 'St. Thomas's Reports,' vol. v. pp. 18, 19.

The rates of mortality of the most eminent French physicians are stated as follows:

Louis	30·8 per cent.
Andral	55·4 ,,
Chomel	32 ,,

For mortality of some of the London hospitals, see p. 139.

[1] It is true, of course, that these sources of error tend to disappear as the numbers compared are increased, so that a great disparity in the death-rate of two large multitudes ever so mixed would tell at last in favour of one treatment rather than the other. Only, as will be seen, we never get the numbers, or anything approaching the numbers.

administered by an intelligent hand, and altered and adjusted to suit the changing circumstances of the case. If matters go well it may be nearly followed; if they go ill it will be variously modified or wholly abandoned. In this adaptation of means to ends, with a single view to the individual life, no inflexible method can find a place. Granted that the material and the discernment could be found for the correct grouping of parallel cases in sufficient numbers, where shall we find the physician who would pursue without deviation a prescribed course of treatment?

Now, if from considering the kind of evidence that we might justly require we turn for a moment to the kind that is actually provided, what do we find? Some scattered tables (in no case dealing with large numbers), without classification of age, details of physical signs, or indication of any kind by which to judge of the severity of the disease. No more, indeed, than a statement of numbers, with the assertion that a death-rate, in many instances large, has been so far reduced by a particular method of blood-letting or the use of a certain drug.

From such material the conclusions to be drawn are of a very general kind. It would appear, for instance, upon this evidence, taken in the gross, that since the time pneumonia was first rigidly defined up to the present day, its rate of mortality on the whole has been decreasing. It would seem, further, that of all the circumstances affecting this rate the time of life is the most potent; the old are likely to die, the young to recover. It will be found, too, as we shall see, that a decline in the mortality of pneumonia is coincident or nearly coincident with the decline or abandonment of the practice of bleeding; a fact, it must be remembered, which of itself witnesses nothing as to the relation between the disease and its supposed remedy.

With the knowledge of this lessened fatality of pneumonia, whether dependent or not upon altered practice, it is quite certain that we shall not willingly revert to systems of treatment which, however appropriate to their own time, concurred with a higher rate of mortality than that we observe now. The history of these old methods, therefore, need not detain us. 'Hujus morbi curatio,' said Sydenham, enforcing the doctrines of Celsus[1] and Galen, 'in repetita venæsectione fere tota est.' Blood-letting was the only hope in pneumonia, and the times and manner of it were minutely described. The practice of England, however, was exceeded by Italy. A century ago, as Grisolle tells us, the elder Frank, in his public practice at Pavia, bled his patients twelve or fifteen times apiece. 'The mortality,' he adds, 'was enormous.' Some years later (at the end, that is, of the last century), whether in mere revulsion from that extreme, or because pneumonia ceased to exhibit the same virulence, bleeding was for the time almost abandoned. In Vienna especially it was rare for a single bleeding to be practised. Such forbearance did not last long, yet from time to time practitioners appeared (whom Laennec called 'heretics') who either abstained from bleeding altogether or employed it but sparingly.

But the extreme of bleeding was reserved for the present century. At the time when the allegation of Sydenham was already beginning to be questioned, there arose in Italy a school which not only reasserted the supremacy of venesection, but combined with it an ally believed to be as effectual as itself. Remorseless bleeding is associated with the names of Tommasini and Rasori.[2] In Parma, Bologna and Milan the pupils of these professors carried out their masters' teaching by removing from the subjects of pneumonia some ten pounds of their vital fluid. The plan was to bleed morning, noon, and evening for the first day, taking two pounds of blood ; for the second day the same amount was obtained in two bleedings ; the rest was got by repeating the operation daily, larger or smaller quantities being withdrawn according to circumstances.

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[1] Celsus, however, must not be represented as an indiscriminate bleeder. Speaking of pneumonia (lib. iv. cap. vii.), he says: 'Oportet, si satis validæ vires sunt, sanguinem mittere; sin minores, cucurbitulas sine ferro præcordiis admovere.'

[2] 'Tommas. dell' Inflammazione,' 1820–7, Pisa; Rasori, 'Teoria della Flogosi,' 1837, Milano.

With this draining of blood Rasori learnt to combine considerable doses of tartar emetic. It was he who discovered the possibility of obtaining for this drug a 'tolerance' on the part of the system, or, more truly, of the stomach. By continued use it appeared that larger and larger quantities could be swallowed and retained. That such 'tolerance' was not without sacrifice is rendered probable from the unexpected occurrence of death in some instances of not severe pneumonia after supposed cure, and in the course of convalescence. The rate of mortality by this treatment of bleeding and antimony was stated to be about 10 per cent., a result naturally quoted in its favour, and which we must therefore suppose to have compared favourably with the results obtained at the same period under less vigorous treatment. It was alleged especially, though the evidence is obviously insufficient, that by this method hepatisation was prevented.

To this practice by routine and rule of thumb, dealing with the disease as a separate entity with little regard to the state of the patient, succeeded the wiser counsels of the French school, of which Chomel[1] and Andral[2] were the chief expositors. These authors, fully persuaded of the propriety of blood-letting in general and of its special efficacy in pneumonia, objected nevertheless to the stated bleedings of Sydenham and to all rigid rules in reference to a practice which, as they thought, should rather be regulated by the circumstances of the particular case. By this time the teaching of Laennec had directed attention (as we may think now too exclusively) to the physical signs within the chest. It was learnt that in pneumonia the lung underwent a series of changes little influenced by therapeutical measures. In the hope of mitigation and not of cure, Chomel and Andral and Grisolle bled and bled repeatedly, but only when the patient was strong and the disease new; they admitted many exceptions to that treatment, and recognised in particular certain epidemic constitutions where venesection was best dispensed with.

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[1] Chomel, 'Leçons de Clinique Médicale,' tome 3, 1834–40.

[2] Andral, 'Clinique Médicale,' Spillan's trans., 1836.

This reasonable teaching was rudely broken in upon by the imperative assertions of Bouillaud. With a singular confidence, certain to be popular and likely to be wrong, this physician, as lately as 1835,[1] put forward, for universal adoption, a treatment which went by the name of the jugulant method, or method of bleeding 'blow upon blow.' By this plan pneumonia was to be strangled almost in its birth. Three days of blood-draining would destroy 'most pneumonias of the first degree about the third day of treatment.' Yet it does not appear that Bouillaud in Paris removed more blood or so much as did Rasori at Pavia. He was commonly content with five pounds of that fluid, and Rasori often took ten. But while Rasori explicitly recognises the fact that the disease 'has always a necessary course,' Bouillaud regarded it as within his personal control; by his strangling method he could suddenly arrest it. The fact that in 102 cases so treated by himself from 1831 to 1834 it was not so arrested in 12, since that number died (a mortality greater than Rasori's), would seem inconsistent with that assertion. The professor insists, however, that these results were, in fact, favourable as compared with the death-rate of the period, and that the fatality was made up of cases too extensive to yield to his strangulation, or so far advanced as to kill their subjects before there was time to put his method fully in practice.[1] Bouillaud's method was, in fact, an imitation of Rasori's. Its pretensions were greater, but, so far as comparison may be instituted, its performance was less. It had indeed something of anachronism. The time was past when the proposal to reduce treatment to a set of formulæ could escape criticism. Bouillaud was attacked accordingly by Grisolle, who only too laboriously and with the vigour of a contemporary denied that this 'blow upon blow' treatment either reduced the duration or diminished the fatality of the malady.

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[1] 'Journal Hebdomadaire des Sciences Médicales,' 1835; Grisolle, *loc. cit.* p. 585.

Meanwhile the most trusted practitioners of our own country, though they might differ in matters of detail, were united in endorsing the statement of Sydenham that in pneumonia safety was to be sought by means of the lancet. While Bouillaud in Paris was pretending to crush it by repeated blows, Gregory[2] in Edinburgh was teaching that with bleeding and water gruel all other help might be dispensed with. Louis indeed had already expressed doubt as to whether the efficacy of bleeding were not confined to an early period of the disease, but his opinion was only quoted in England[3] that the inexperienced might be cautioned against it.[4]

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[1] It is hardly necessary here to indicate the details of this system, which comprehended strict rules for the conduct of each day. Between the daily bleedings thirty leeches were to be applied to the painful side. The slight cases would yield to three bleedings, the graver might require seven or eight or nine in addition to the leeching. If on the third day—contrary to the asserted rule—the disease still survived, general bleeding (of three or four palettes) was to be at once repeated. In the rare instance of still further resistance it was usually deemed best to abandon blood-letting and apply a large blister.

[2] See Watson's 'Lectures on Medicine,' vol. ii. art. 'Pneumonia.'

[3] 'I advert to his opinion,' said Sir Thomas Watson, lecturing at King's College in 1838, and alluding to M. Louis, 'merely to caution you against being misled by it, as you might otherwise be, considering his well-merited reputation as an exact and faithful observer.'

[4] 'Recherches sur les effets de la Saignée,' &c. Paris, 1835.

The doctrine of that day was in the main that of Laennec. It was precise and definite, and obtained a weight of sanction hardly attainable upon any therapeutical question at the present time. Bleeding being the great remedy, the earlier it was practised the greater and the more certain the relief. Its amount was to be governed in each case by the impression produced upon the system, measured partly by the sensations of the patient, partly by the greater softness and fulness of the pulse, or, these signs not appearing, by the approach of syncope. The effect of a first bleeding in removing pain and dyspnœa was to be carefully watched. Usually a return of these symptoms would necessitate its repetition within four or five hours. Supplementing such treatment, cupping, or the application to the chest of a large number of leeches, was recommended. The diet was to be 'low and unstimulating.'

As the disease advanced, so, it was believed, did the abstraction of blood become less efficacious, but even with a solid lung bleeding was of use as 'tending to prevent the extension of the inflammatory process.' Yet a time might come when the shock of bleeding could no longer be borne, and other less powerful antiphlogistic remedies had to be substituted. Of these tartar emetic was appropriate for the stage preceding hepatisation, and mercury for hepatisation itself. The first, in frequent doses of a third of a grain (guarded at first by laudanum and increased, if necessary, up to two grains per hour), would often put an end to dyspnœa and allay any '*disposition to rekindle*' on the part of the inflammation; the second (blue pill or calomel, in small and repeated doses of a grain or two), was to be so administered as to render the gums tender as quickly as possible. Even should the lung remain solid under this treatment, it was still to be persevered with until the pulse and face betokened the approach of death.

Such was the teaching of the schools in London and Edinburgh fifty years ago.

M. Grisolle in the edition of his well-known work published in 1841[1] somewhat modified these views. Though favourable still to blood-letting, he for the first time fully recognised in distinct terms what Rasori had already expressed in words but denied in practice, namely, the natural course which the disease was destined to fulfil, and which, whether grave or not, the conduct of the physician could not appreciably alter.

Thus, although bleeding in some sort was during a long period the universal remedy for inflammation, and especially for lung inflammation, it must be noticed that it was not practised always to the same extent or to accomplish the same ends. As medicine advanced the same weapon was put in succession to several uses. Bouillaud, by a strange and mixed figure of speech, strangled pneumonia with the lancet by repeated blows; Rasori, not less prodigal of blood, confessed himself unable to accomplish so much; while both Chomel and Grisolle clearly recognised that, whatever the method employed, pneumonia had its period of rise and decline notwithstanding. It is the same with tartar emetic. The one drug sufficed to give effect to many theories. Between the 'toleration' and high doses of Rasori and the small diaphoretic doses of more modern practice there is expressed a world of gradation or revolution in medical opinion as to the right treatment of the disease.

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[1] Grisolle gives an analysis of 50 cases of pneumonia treated by bleeding at the first stage of the disease, and 182 so treated at the second stage. The bleedings were practised from one to five times, upon no uniform plan, more or less often, according as the patient responded to it early or late. Hence those that died lost most blood, probably from two to four pounds. Of the 50 bled at the first stage 5 died; of the 182 bled at the second, 32; a mortality much less than Andral's, and in the circumstances of the time and of the cases not unfavourable. M. Grisolle concluded from personal observation that bleeding, however practised and however accompanied, had no power to arrest the disease. He thought that it brought about a general improvement in the patient's condition, but with no corresponding change in his physical state. The benefit to be expected from bleeding is, in his view, that it disposes the economy to receive other therapeutic agents. Local bleeding, he believed, had a value of its own in relieving the pain of stitch.

Yet while it remained the orthodox teaching up to 1847 (the date of Sir T. Watson's 3rd edition), that general bleeding was necessary in pneumonia, and that the quantity of blood taken should always be large, already there were signs of schism.[1] Louis had ventured to doubt the use of venesection except it could be practised early. Grisolle had convinced himself by the contemplation of Bouillaud that blood-letting did not effect all that was alleged of it. In the meantime attention had been directed to the virtues of tartar emetic given in large doses after the method of Rasori, and, as in the case of every drug brought prominently forward for a new use, assertions were now made in regard to antimony which we may well believe to be extravagant. Yet it was not unreasonable to hope that tartar emetic might at least aid, and in part spare, the work of the lancet. Already Laennec had announced that with moderate bleeding and tartarised antimony his success had been greater than with bleeding alone or any other method. Louis had recorded in detail the sudden relief that followed large doses of the same drug in critical cases which had been repeatedly bled without benefit, while Trousseau, subscribing to the same treatment, had met with but two deaths in 58 persons in 1831 and 1832.[2]

Influenced mainly by such testimony, our own English practice drifted into a middle course, using bleeding a little and antimony a little, with a much diminished trust in the efficacy of both. And now, with the abandonment of heroic treatment, there concurred a much lower mortality. Pneumonia ceased to be the formidable disease it had once been. The tendency to recover became more conspicuous than the tendency to die; the mortality, so to speak, became tolerable.

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[1] See Balfour, 'Edinburgh Medical Journal,' 1858, p. 214 *et seq.*, 'Hæmatophobia.'

[2] He admitted a less success by the same means in 1839, and believed that owing to the circumstances of the time bleeding became necessary. Yet of the effect of antimonials he still speaks (as indeed does Grisolle himself) in terms so high that he foresees that a future generation will tax him with exaggeration. Nothing is more remarkable than the rapidity of recovery. 'There is no convalescence. Three days suffice sometimes to bring back the patient from the gate of the grave to a state of health.' Little did the author expect, so grievously had drug interference disguised the real character of pneumonia, that these words fairly describe the natural course of the disease in many cases under our modern treatment.

We cannot doubt (what, indeed, the literature of epidemics strikingly illustrates) that the mere death-rate of a disease will influence its treatment beyond what reason can justify. The remedy must be made to appear to the public eye proportionate to the malady. As the latter becomes less destructive a relaxed vigour in treatment is likely to ensue, which, though really the consequence, may easily be mistaken for the cause of the amendment. The publication, therefore, at this particular epoch of the disease of certain statistics (as of Skoda in 1843–6, of the homœopathists in Vienna, of Barthez in Paris, and more recently of the late Dr. Hughes Bennett in Edinburgh), proving that the subjects of pneumonia might survive without medical interference, was taken to prove that the affection did best when left to itself.[1]

The history of this change in opinion is to be sought in periodical medical literature between 1847, when Dr. Balfour called attention to the treatment of Skoda,[2] and 1866, when Dr. Markham, in the Gulstonian Lectures before the College of Physicians, discussed the reality of the asserted change of type in disease. Of the new views perhaps Skoda was the pioneer. Losing faith in active depletion, he confined its employment to the period preceding hepatisation, and then used it only when the fever was high and dyspnœa urgent. In his treatment by corrosive sublimate (which he gave 'to reduce the plasticity of the blood'), he had a mortality of 1 in 7 with 392 patients, a result 'particularly favourable' at the time, especially as compared with the Edinburgh Infirmary (1839–44), where the death-rate upon the old treatment was, as it seems, unprecedentedly high, namely, 1 in 2·78.[1] Skoda, however, did not so much contend for a larger number of patients saved, as for the more speedy recovery of those that got well.

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[1] See tabular statement, p. 394.

[2] Balfour on the Practice of Skoda, 'Ed. Med. and Surg. Journal,' 1847, pp. 397, 398. Dr. Markham's lectures are published in a pamphlet.

The propriety of non-interference was further shown by the statistics published about the same time by Dr. Fleischmann, showing a mortality of less than 6 per cent. obtained through homœopathy. Some doubt has been cast upon the accuracy of these figures, which provoked at the time much angry controversy. It would be profitless to discuss them now; right or wrong, they had no small share in directing attention to the possibility of abandoning active remedies for pneumonia, and thus helped to lay the foundation of the so-called 'expectant' method.

Acting upon this hint, Dietl, in 1844–6, employed what he called a dietetic treatment in 189 cases, with a mortality of 7·4 per cent. He was less fortunate in a second series of 750 cases, where, nevertheless, the death-rate of 9·2 was more favourable than either Rasori's with high doses of antimony, or Bouillaud's with his repeated blows. There are no means now of analysing Dietl's results. As is usual, the treatment he pursued was less successful in the hands of his successors. It accomplished no further result than to encourage further trials. There was at least evidence enough to show that pneumonia might run its course without medicine, and that the result of non-interference, as compared with the most active depletion, was not necessarily disastrous.

But the testimony did not end here; it was soon admitted to be the *habit* of pneumonia to recover. With children, at all events, from two years old up to fifteen, it was asserted to be very rarely fatal. Out of 212 such patients Barthez reported that he lost but 2, this low mortality arising, not from any measures of his, but from the benignant nature of the disease. Similar testimony was borne by England and Scotland. Never, under any system, homœopathic, allopathic, jugulant, or expectant, had such results been made public. Was this happy change due to an alteration in the nature of pneumonia itself, or in the current nomenclature, or to the fact that the methods heretofore supposed to be beneficial were in fact injurious?

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[1] Bennett's 'Restorative Treatment of Pneumonia,' p. 41.

To that question, difficult as it may seem, there were some who ventured to return a very positive answer. That pneumonia had never been so little fatal as in a certain period during which active medication had been withheld, was given in the facts. That to abstain from active medication, of whatever kind, was the best treatment for pneumonia, was announced as the just conclusion. And soon, as though it were a corollary to that proposition, it was further declared that in this particular affection, while drugs were useless and worse, food was of exceptional benefit.

Now it is not more reasonable, *à priori*, to assert that pneumonia may be conducted to a safe issue and have its mortality annulled by a uniform practice of feeding, than it would be to maintain that its progress may be arrested by the uniform draining of blood. It is even less reasonable. For while blood may be abstracted from every living body, the power to swallow food, and, much more, to retain and assimilate it, is wont to disappear very early in acute illness, and when the needs of the body are the sorest. The treatment is admirable for the favourable cases, but the very terms of its statement imply that its application will be limited to these. 'Good nutrition' and 'exalted vital force' are likely to be associated with such examples of pneumonia as recover ; for the rest, the mere insertion of food into the mouth does not insure nutrition or promote vital force. The ability to receive food as such is absent.

In speaking of the restorative method, therefore, it is necessary to distinguish between actual statistics and the arguments used to support them. The one must not be allowed to supplement the other. Any such plea as that small numbers may suffice because the truth of the principle is self-evident must be rejected. The numbers must stand by themselves and the arguments by themselves.

Reading now the 129 cases adduced by the late Dr. Hughes Bennett in support of his method, but without his commentary,[1] this is what we find. The average age of the patients was 30½ ; 125 recovered and 4 died, a percentage (excepting Barthez with children) by far the smallest we have yet met with ; 88 of the patients were under 40, 24 between 40 and 50, 13 exceeded 50, 2 of them being between 60 and 80. Of the 125 that recovered, 105 were simple and 20 'complicated.' Of the former, 79 were single and 26 double. The duration of the disease, on the average, was 14¼ days.

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[1] See Dr. Hughes Bennett on the 'Restorative Treatment of Pneumonia,' pp. 45–50. 'This treatment,' says he, 'may be said to render the mortality in true cases of pneumonia *nil*.' 'The formation of pus is the normal and necessary transformation of the solid exudation, whereby it is broken up and caused to be absorbed.' Pus cells, it is explained, must be regarded as living growths, and 'as such require an excess of blood, good nutrition, and exalted vital force to hurry on their development and carry them successfully through the natural stages of their existence.'

The following extract gives Dr. Bennett's method in his own words: 'To further the necessary changes which the exudation must undergo I content myself with giving salines in small doses during the period of febrile excitement, with a view of diminishing the viscosity of the blood. At the commencement of the treatment I order as much beef-tea, milk, and other nutrients as can be taken, and, as soon as the pulse becomes soft, solid food and from four to eight ounces of wine daily. As the period of crisis approaches I give a diuretic, consisting of half a drachm of nitrous ether, and sometimes ten minims of colchicum wine, three times daily, to favour excretion of urates. . . . It differs entirely from the expectant treatment in the care which is taken to nourish the weakened frame from the beginning,' &c., &c., *loc. cit.* p. 51. It is added, moreover, that by this treatment (1) the blood is rendered less viscous by salines ; (2) the system is fed by means of wine ; (3) urates are excreted by means of colchicum and nitrous ether.

These numbers (given by Dr. Bennett with much detail) are sufficient to show beyond doubt that, in respect both of duration and mortality, the successful issue of his cases is exceptional as compared with other statistics.

It is to be regretted, therefore, that results so satisfactory cannot in strictness be adduced to support any treatment in particular unless it be the giving of alcohol. These tables do not even illustrate, for good or bad, the effect of any single treatment, 'restorative' or otherwise. Thus, taking the 20 worst cases, or at least, those that appear to be the worst, upon the evidence of pulse, respiration, and extent of lung engaged, it is only in 7 that the 'nutrient' system was applied. The other 13 were treated with bleeding, or antimony, or laxatives, or salines. The worst cases got alcohol, some of them in considerable quantity. One of these is stated to have been 'saved' by half-ounce doses of wine every half-hour. If the enumeration had been framed upon Grisolle's plan of excluding those that would almost certainly recover anyhow, it would have been reduced to 53, for only that number had on admission a pulse over 100; if it had excluded those who were not brought under Dr. Bennett's treatment till after six days' illness, it would have been reduced to 76; if it had included those only upon whom the nutrient system was in fact fully tried along with wine, but with no active drugging, it would have comprised but 46. Of the treatment by nutrients apart from wine there is hardly a case, certainly not a grave case. What the table shows chiefly is the treatment by alcohol, in moderate quantity for the most part, but indefinitely increased when occasion seems to require it. The drug which, after alcohol, is most conspicuous is antimony. Such statistics illustrate only what was said at the beginning, that the course of treatment, inasmuch as it follows the course of the disease, is seldom, in fact, uniform.

In the list following (*which is a verbatim extract*) we have included nearly all the crucial cases of Dr. Bennett's 129.

No.		First seen after rigor	Pulse	Respirations	Treatment	Observations
		days				
Men.	21	5	130	30	¼ grain antimony every hour; afterwards every 2 hours	Rapid recovery
	22	5	148	56	Antimony ⅙ grain every 3 hours; afterwards nutrients	Convalescence not determined
	24	6	120	44	⅓ grain antimony every 2 hours, diuretics ℥vj., wine, and nutrients	Of intemperate habits
	36	7	120	40	Salines, diuretics, c. colchicum wine ℥iv., and nutrients	A debilitated intemperate man
	37	1	120	36	Salines, blister, nutrients	A case of pleurisy, pneumonia intercurring
	48	6	120	48	Wine ℥iv. daily, liquid nutrients *ad lib.*, slight salines	Recovered rapidly
	51	2	120	46	Salines, strong beef-tea, wine ℥iv.	—
	61	5	104	56	Salines, slight diuretics, wine ℥vi., nutrients	Very severe case
	75	3	120	66	Nutrients, wine ℥vj. . .	Rapid recovery
Women.	86	6	120	dyspnœa	Bled to ℥xij. on admission, ½ grain antimony every 2 hours	Record defective
	89	8	132	hurried	Digitalis, laxatives, 3 leeches to side	Great exhaustion and unusual action of heart
	95	5	120	hurried	1 grain antimony every 2 hours, afterwards 12 leeches, wine ℥vj., blisters	Very weak after subsidence of fever
	96	1	120	36	Salines, 8 leeches, and afterwards blisters	A simple pneumonia
	105	7	120	2	Antimony ⅓ grain and diuretics	—

No.		First seen after rigor	Pulse	Respirations	Treatment	Observations
		days				
WOMEN.	106	2	130	32 to 36	Antimony $\frac{1}{3}$ grain every third hour; after $\frac{1}{4}$ grain every fourth hour	—
	107	5	112	40	$\frac{1}{3}$ grain antimony, 10 leeches, and a blister	—
	111	11	120	urg'nt dyspnœa	Wine, nutrients? ℥vij. (sic.)	—
	114	1	120	dyspnœa	Salines, nutrients, wine ℥iv.	Complicated with bronchitis and phthisis
	116	3	150	56	At first salines; afterwards diuretics, ℥ss. wine every half-hour, new milk, and strong beef-tea	*Saved by ℥ss. wine every half-hour*
	124	8	120	65	Salines, wine ℥iv., nutrients	—

These are, it will be seen, but twenty cases out of more than a hundred, but for testing the point at issue they are *the* cases upon which the efficacy of the treatment is to be judged. And what is that treatment? With no less than eight of the twenty it is antimony; with the others it is wine, which is given in larger or smaller quantity according to individual need, sometimes in aid of the 'nutrients,' and sometimes for the immediate saving of life.

Similar objections would apply to the treatment records of other physicians. Thus Dr. Waters, of Liverpool, tabulates seventy-seven cases. From a careful analysis of these we learn that at least twenty-three were admitted on and after the eighth day. Fifty-four only can be taken as fair illustrations of the hospital treatment. Of these twenty-six, as far as can be judged from the details given, were serious cases. One died. Of cases of simple pneumonia, subjected to Dr. Waters' treatment within a week of commencing illness, we cannot reckon more than twenty-three. Of these nine were severe. Three of the number were treated with antimony, and six with wine or brandy.[1]

It does not appear, indeed, that the table is intended to illustrate the success of a particular treatment, but rather to show the usual course and small mortality of the disease. The patients are too few and are seen too late to serve for more than this, while the remedies used are simply adapted to the particular wants of the individual. Beef-tea is administered as such, as the common food of the sick ; salines are used in obedience to a common routine, apart from the 'viscosity of the blood,' and, in the important matter of direct stimulation, alcohol is given or withheld as the circumstances seem to require ; in the last resort, as with Dr. Bennett, it is trusted in entirely as the sole stay and hope of the patient.

Thus the question as to the actual numbers which might fairly be regarded as sufficient to illustrate the effect of a particular course of treatment does not in fact arise, since in such numbers as we get, no particular course of treatment is actually put in practice.

With such evidence as to the value of numerical statements we insert for what it is worth the following Table, representing the results of treatment under various systems, homœopathic, non-homœopathic, expectant, &c.

This enumeration may be trusted to speak for itself. All that need be said of it has reference to Grisolle, whose elaborate statistics, whatever their real value, cannot fairly be treated in this summary way.

The value of *tartar emetic* was tried by this physician upon 154 persons, classed as follows : forty-four were treated with it alone in half-hourly or hourly *emetic* doses ; eighty (after the manner of Laennec) were bled first and given antimony

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[1] See Dr. Waters 'On Diseases of the Chest,' art. Pneumonia.

TABULAR STATEMENT OF RESULTS OF TREATMENT IN PNEUMONIA (FROM DR. ROGERS' 'THERAPEUTICS')

	Where and by whom Treated	No. of Patients	No. of Deaths	Average Mortality per cent.
	Homœopathic Treatment:			
	Fleischmann (7 successive years) . .	239	14	5·85
	Eidherr (7 successive years) . . .	—	—	7·22
	Homœopathic section of Leopoldstadt Hospital (6 successive years)	94	9	9·57
	Do. Wurmb and Caspar, 1850 . . .	24	3	12·55
	Tessier	41	3	7·3
	Non-Homœopathic:			
1st Group	Hegelé, hydropathic treatment . .	40	None	—
	Barthez, expectant treatment (children) .	212	2	0·94
	Ziemssen ,, ,, . . .	201	7	3·48
	Dietl (1st series) expectant treatment .	189	14	7·4
	,, (2nd series) ,, ,, .	750	69	9·2
2nd Group	Bennett, restorative treatment . .	129	4	3·1
	Kissel (acet. ferri or acet. cupri) . .	112	5	4·4
	Sauer (sulph. cupri)	56	3	5·35
	Edinburgh Infirmary (1865) . . .	36	3	8·33
	Allopathic section of Leopoldstadt Hospital (6 successive years)	104	13	12·50
	General Hospital, Vienna (10 successive years)	—	—	18·28
	Huss[1] (8 successive years), 2nd series .	—	—	10·21
3rd Group	Lebert	—	—	7·3
	Huss (8 successive years), 1st series .	—	—	11·50
	Grisolle	—	—	16
	Louis	—	—	30

The above groups are defined as follows:

1st Group.—That in which no bleeding and few or no drugs were employed.

2nd Group.—That in which medicine was more employed and bleeding sparingly.

3rd Group.—That in which both medicine and bleeding were actively employed.

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[1] Huss's statistics extend from 1840 to 1855, and include 2,616 cases, with a total mortality of 281, or 10·74 per cent., the largest numbers we have to deal with. The yearly rates, however, are very various. In 1843, 14·19 per cent. died; in 1851, only 6·19—an instructive observation in reference to the conclusions to be drawn from statistics.

subsequently and in place of further bleeding; the remaining thirty were given antimony as a *pis aller* at a late period of their illness, and because further bleeding was deemed impracticable. Of the first class Grisolle lost six, or one in 7⅓, but *none of these commenced the treatment till after the fifth day.* Of the second class ten died, or one in eight; the treatment with them was not begun till the sixth or seventh day. Of the third class eighteen died out of the thirty. Of the twelve who recovered, nine 'in the greatest peril' improved rapidly under the antimony. Of the whole only twelve absolutely and unfailingly 'tolerated' the remedy, yet in all 'it was evident from the diminished *malaise* that it should be persisted with.' The patients improved with a rapidity unobserved in any other treatment, a statement corroborated, as we have seen, by Trousseau.[1]

Turning to the evidence for and against the use of special drugs, there is little recorded in reference to such agents (with this exception of antimony) that need detain us. The salts of iron and of copper, acetate of lead, veratria, alkalies, chloroform inhalation, and other things have, indeed, been hypothetically recommended, and statistical evidence has been adduced in favour of some of them. Thus Kissel in 1848–50 with acetate of iron and acetate of copper lost 4 cases out of 93, or 4·4 per cent., 70 *of these being under* 20 *years old.* Sauer with sulphate of copper lost 3 out of 56, or 5½ per cent. Hegelé in 1848–9 with hydropathy, *the patients being young*, lost none out of 40, or nothing per cent. It is idle to accumulate naked figures like these, nor is it too much to say that there is no published account respecting any of the drugs named entitled to consideration.

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[1] Yet the effect of antimony was not in itself pleasing. Not rarely it produced in the first twenty-four hours from ten to fifteen vomitings, and from twenty to thirty stools, the latter being always in excess of the former; the treatment was, indeed, regulated so to act. Nevertheless, in the concurrent testimony of two independent observers, the general improvement following these truly drastic measures was so obvious as to suggest the figure of a man snatched from the grave.

It may be urged, however, that there are other modes of estimating the value of drugs besides the numerical; that, for example, a medicine may be rationally selected on the ground of its ascertained physiological action, even though statistical results in reference to the particular disease are not obtainable. In this view digitalis, aconite, quinine, veratrum, ether and chloroform inhalation have been employed in pneumonia in virtue of some influence they are believed to possess, whether in moderating the pulse, reducing the body temperature, producing diaphoresis, or easing pain.

Digitalis, indeed, had already been largely tried by Wunderlich, Ziemssen, Liebermeister, and others, to reduce the pyrexia of typhoid. Bleuler making trial of this same drug in pneumonia got the high mortality of 21 per cent., as against 14·5 per cent., the mortality without it. He did not himself note any effect of the medicine in cutting short the inflammation, but remarks upon the early period of defervescence in his own cases, namely, from the fifth to the seventh day. Bleuler gave as much as half a drachm of digitalis daily in powder, and elicited in some instances the poisonous effects of the drug. Liebermeister was led by experience to confine the use of digitalis as an antipyretic to cases in which the pulse is not unusually high and no pronounced symptoms of cardiac weakness exist. He regards it as less efficacious than quinine, and would employ it only at the commencement of pneumonia. His dose was from 10 to 20 grains in powder or pill every twenty-four or thirty-six hours.[1] M. Piory-Sancerotti gave foxglove in 35 cases of pneumonia, and concluded thence that 'as an antiphlogistic it was less rapid but more durable than leeches.' Furthermore, the use of digitalis in pneumonia has the approval of Niemeyer, an approval which Stillé insists that there is no direct evidence to justify.

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[1] Ziemssen's 'Handbook of Therapeutics,' vol. ii. pp. 88, 91, &c.

Veratrum viride and veratrine have been recommended in pneumonia to reduce pyrexia. Dr. Kiemann gave the former in 40 cases, 5 of whom died. The action of the drug as an antipyretic was marked and invariable. There is other testimony by Drasche and Vulment to the same effect, though the former adds that veratrum rather postpones the process of resolution.[1]

Veratria, on the other hand, upon the evidence of Kocher, who had large experience of the drug, shortens the duration of this process. It reduces the pulse and temperature, and sometimes appears to cut short the disease and prevent consolidation. The author alludes, however, to the dangerous depression produced sometimes by veratrine, and believes that it cannot be safely given in larger quantity than one-twentieth of a grain at intervals of from one to two hours, until an obvious impression is made on the pulse or the temperature. The good results obtainable from veratrine may be promoted, according to the same authority, in severe cases by bleeding. Other authors have made use of this agent and generally commend it, as Aran, Vogt, and Trousseau. Its power to reduce temporarily the frequency of the pulse is pretty well established, but it is agreed that its effects are not constant, that it has to be used with great caution, and is apt to cause diarrhœa and vomiting. Liebermeister believes that cardiac weakness contraindicates the use of veratrine, and admits that in recent years he has given up its use in the belief that its possible indications may be better met by means of salicylic acid.

Aconite has been much extolled as a reducer of body temperature. As regards its use in pneumonia Dr. Phillips[2] records that in 9 cases under his own observation the temperature fell in from 3 to 6 days and the pulse with it. From 11 other cases he judges that aconite has no effect in removing consolidation, but 'controls and removes the tendency to spreading of the congestion.' Dr. Wilson Fox gave aconite in one or two cases, but could not observe any effect produced by it on the temperature; while Dr. Anstie, on the contrary, used to regard it as the most certain and reliable of diaphoretics.

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[1] Phillips, 'Materia Medica,' art. Veratrum.

[2] *Loc. cit.* p. 7.

Alkalies have been commended in pneumonia. By the use of bicarbonate of potash in doses of from 5 to 30 grains, largely diluted, Dr. John Popham[1] believed that a sedative effect was produced, and that the 'physical signs became resolved.' As an instructive commentary upon this statement, and upon the asserted power of alkalies to prevent hepatisation, cases may be referred to where pneumonia of fatal severity has succeeded acute rheumatism, notwithstanding that the patient was at the time taking alkali in high doses.[2]

In its action as an antipyretic careful and extensive observations have been made upon quinine by Vogt, Courvoisier, Liebermeister, and others,[3] but they do not especially refer to pneumonia. On the whole, the very high estimate placed upon quinine as a reducer of temperature in Germany is not borne out by British observers. Thus, in 1870 a Committee of the Clinical Society made an inquiry into the action of the drug in the pyrexia of various diseases. As regards pneumonia only a single instance appears. Ten grains of quinia were given on the morning of the fourth day during the pyrexial remission. A reduction of temperature followed amounting to 2° of Fahrenheit. The 'effect' lasted 24 hours, the pulse and respiration being but slightly reduced. After 30 hours the temperature 'returned to its ante-quinine range.' In several other diseases, though the pulse fell with the quinine, the respiration was quickened. The Committee adds, as respects quinia in its action in reducing temperature, and the statement in our belief is equally applicable to other drugs: 'There was no conclusive evidence that the quinia favourably influenced the duration or course of any of the diseases in which it was administered.'

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[1] 'Brit. Med. Journ.' Dec. 28, 1867.

[2] See p. 214, Chap. XI.

[3] Ziemssen's 'Handbook of Therapeutics,' ii. p. 66 *et seq.*

Those authors who attach high value to inhalation in the treatment of lung diseases have not overlooked pneumonia as a condition sometimes favourable for its employment. Prof. Oertel in his elaborate work on 'Respiratory Therapeutics'[1] quotes the opinion of Baumgartner in favour of ether and chloroform inhalations in inflammation and other affections of the lung. Such inhalations, it is asserted, relieve the feeling of constriction and 'the stitch in the side,' while they diminish expectoration and sleeplessness. The cough, however, it is added, is sometimes aggravated in the early stage of ether inhalation. Several German authorities are quoted by Prof. Oertel in addition to his own experience, favouring this treatment. It is to be applied in the later stages of the inflammation when respiration, owing to pleuritic stitch, is frequent, short and irregular, secretion suppressed, and the surface cyanosed. The inhalations are administered three to four or six times a day up to commencing narcosis, with the general result that the symptoms just mentioned are relieved, bronchial secretion becomes more or less copious, and cyanosis decreases to a remarkable degree. The mode of administration is simply to hold before the mouth and nose a handkerchief with from ten to twelve drops of chloroform, in such manner as to permit atmospheric air to be breathed along with the anæsthetic. The contraindications to this treatment are stated to be 'tendency to headache, giddiness, and rapid stupor, extensive engorgement of both lungs, and general prostration.' It is added, moreover, that 'attempts to treat pneumonia from the first with chloroform inhalations have naturally led to no result, as its influence is powerless to arrest the inflammation itself,' and further that 'the high fever, uneasy decubitus, and above all the extreme vulnerability and irritability of the inflamed lung contraindicate every mode of treatment that could excite any increased inflammatory reaction.'

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[1] Ziemssen's 'Handbook of Therapeutics,' vol. iii. pp. 141 and 324. Translated by Dr. Burney Yeo.

Of a method of treatment encompassed by such restrictions it is unnecessary to say much. Chloroform inhalation reserved for the later stage of pneumonia, about the fifth or sixth day, 'when the bronchi are filled with secretion, a large portion of the lung rendered useless, and the face cyanosed,' is at the same time, we are told, contraindicated by general prostration, rapid stupor, and extensive engorgement of both lungs. Thus the symptoms that indicate its use are closely associated with those that indicate its danger. If chloroform inhalation is to be restricted to patients in the fifth or sixth days of pneumonia, involving a large portion of lung together with general bronchitis and already cyanosed, yet without prostration and in no near danger of stupor, then its use must be confined to a very limited class of cases, with which we do not profess to be familiar. Prof. Oertel's statement, therefore, that he has 'obtained favourable results in suitable cases,' does not imply that he has used it largely. The well-established physiological action of chloroform would seem to forbid its employment in any acute disease of the respiratory organs.

Of the treatment by bathing and cold packing we shall speak in the next chapters, in connection with other appliances which in our belief are of proved service.

In the foregoing review of the treatment of pneumonia in the past the reader cannot fail to be impressed with the fact that, amid much discrepancy of statement and conflict of opinion, the final issue depends far less upon the particular method of treatment than upon the source whence it comes. The statistics derived from particular 'schools,' or illustrating the practice of some individual who himself expounds it, are always the best : those that laborious compilers construct for themselves, regardless of persons or theories, from general hospital records, are always the worst. There is no other rule that works. Thus, Bouillaud's industrious bleeding is almost as successful as the do-nothing of homœopathy, and both methods are only excelled by the 'nutrient' treatment of the late Dr. Hughes Bennett ; while, on the other hand, the mortality of the great European hospitals, and of eminent physicians in the past, pledged to no system and entering into no competition, is as a rule the largest, or if favourable in one year is disappointing the next.

CHAPTER XX

TREATMENT IN GENERAL

Practical difficulty in estimating results of treatment—Illustration—General indications—Treatment of pleuritic 'stitch'—Anorexia—Constipation—Rules for diet—Temperature and ventilation of room—Treatment of insomnia—Diarrhœa—Routine remedies.

In the preceding chapter it has been shown that all attempts to estimate treatment in pneumonia by a comparison of results are fallacious; that speaking generally, and with some doubtful exceptions, the more active treatment has coincided with the larger mortality; and that modern practice, recognising a natural tendency to recovery, has reserved its remedies for incidental symptoms without seeking to interfere with the course of a disease it is powerless to arrest. Thus the treatment of pneumonia has undergone a change—or rather a series of changes, corresponding with successive views of its pathology. At first the mere name sufficed to suggest a series of remedies designed for its prompt suppression; presently, upon the gradual abandonment of all hope of that sort, the special value of a not less uniform treatment by means of 'nutrients' was insisted on; and now, recognising the limited applicability of any single method, we have come to consider the individual more and the disease less. Each case is treated, so to speak, upon its own merits. Discarding all notion of specific remedies, we yet believe that particular symptoms, of themselves harmful, may be mitigated or removed, and that modern experience has enlarged our resources in this respect, while it is not unreasonable to assume that by thus placing the patient in the best conditions for recovery we do much to save his life.

Thus, while the function of treatment is limited, its place and use are alike obvious. Almost always there are symptoms of more or less gravity according to their subject, time of appearance, and attending circumstances, which require for their removal experience, patience, and contrivance. Herein consists the whole treatment of pneumonia. How far the final result is influenced by such conduct we cannot say for certain. The natural crisis of pneumonia must always obscure any exact estimate of the help the physician renders. A near correspondence in time of the greatest severity of the disease and the period when it reaches its natural term—habitually overlooked, as we have shown, by the old writers—is even now not seldom ignored, and, as a consequence, many methods of treatment get undeserved credit. It is so important that this common source of error should be kept in view that, although we have already given some instances to like effect (Chap. IX, pp. 162, 164), we think it well to commence this chapter with an example forcibly illustrating the difficulty in question.

William E., aged 25, a warehouse porter, a stout and muscular man of temperate habits and good family history, was admitted into the Westminster Hospital December 7, 1880.

On the evening of the 4th, after his usual day's work, he had a severe rigor, which was repeated next day. Along with this he got cough, 'stitch,' and some dyspnœa, obliging him at once to take to bed, where he remained until brought to hospital on the third day of illness.

For some six weeks he had complained of enlarged tonsils, but with no general impairment of health.

On admission aspect was depressed, temperature 104°, pulse 132, full and compressible, tongue moist and red, sputum scanty, viscid, and rust-coloured. Dyspnœa aggravated by severity of pleural pain. There was consolidation at the base of the left lung, but over a limited area. Some coarse bronchial râles were audible over both lungs.

On the following day (the fourth of illness) œgophony, extended dulness, and more distant, yet still distinctly tubular breathing, indicated effusion. The patient was more depressed and had had no sleep whatever, suffering much pain from stitch. R. 44, P. 132, T. (midday) 104·6, urine, sp. gr. 1024, with trace of albumen. No marked deficiency of chlorides. Great difficulty was met with in attempts to feed. Beef-tea was absolutely declined, and the man took only milk and a little mutton broth. Hot fomentation of itself failing, five leeches were applied to the side with fair success in relieving pain. For medicine he had ammoniated saline draughts every four hours. Next day, the fifth of illness reckoning from first rigor, the patient was notably worse, quite sleepless, restless, delirious, breaking into sweats, the lips and ears now distinctly cyanotic; P. 144, R. 44, shallow, T. 103·8.

It was on this fifth day of illness that it was determined to give alcohol, and he took that day four ounces of brandy, together with such small quantities of milk and mutton broth as it was found possible to administer. That night was sleepless and occupied by delirium like the others, a delirium so active as to need a special nurse to keep the man in bed. With a drying tongue and extreme prostration temperature was in descent, and at midnight (the end of the fifth day) was 100°. He was now given, together with the brandy just mentioned, bark and ammonia in place of the saline.

Passing over the next (or sixth) day, during which his condition as to delirium, prostration and utter sleeplessness remained the same, the seventh day found him semi-conscious, passing urine in bed, much cyanosed, hard to recall to himself. Tongue now dry, with sordes collecting; much sweating. T. 101·8, P. 140, R. 46, at time of visit, but these varying much as the day went on.

In this extreme condition he was ordered half an ounce of brandy every half-hour, and thus took 24 ounces in the 24 hours succeeding. Directions were also given that, should increased rate and difficulty of breathing occur, the patient should be bled to 8 ounces. *During the night of this same seventh day the aggravation of his extreme condition actually happened, and preparations for bleeding were made.* But in the interval the urgency became something less, and bleeding was accordingly postponed.

On the following (eighth) day the patient was still delirious and unresponsive, and still passed urine in bed unconsciously. But respirations were less, 34, as well as pulse and temperature, 126 and 99·4 respectively. The physical signs of consolidation had not changed or extended. He had now been five days in hospital entirely without sleep. But on the afternoon of this eighth day at the time of the visit he was observed to be drowsy. The half-hour doses of half an ounce of brandy were therefore suspended. He fell into a sleep—his first for close on a week, speaking literally. And from this sleep he awoke sensible and safe. The change was at once recognised in his aspect and consciousness, and, even through his sleep, the precise hour of this critical change. On the following (or ninth) day it was reported that he had had a quiet night, sleeping well; P. was 100, T. 99·4, and R. 26. Urine (now first obtainable after interval of three days) was without albumen; the man was rational and drowsily answered questions. To be short, on the ninth day redux crepitation was heard, and tubular was exchanged for bronchial breathing. All medicine, including brandy, was now stopped, and by the twelfth day from his first rigor this man was convalescent, almost all signs, whether of pneumonia or bronchitis, having passed away. Complete recovery of strength, however, was, as might be expected, somewhat more tardy than in an ordinary pneumonia, and some pallor and duskiness of face remained. Still, on the seventeenth day from commencement of illness and the ninth from its extremest severity, the patient was up and about almost in his usual health.

This abstract affords an instructive example both of the indications afforded by symptoms, and of the means of estimating the actual influence of treatment. Of the indications, it may be observed that while temperature and pulse are falling there is no other amendment; on the contrary, during three days of declining pyrexia all the worst symptoms—prostration, unconsciousness, delirium—are accumulating. Even when these symptoms suddenly subsided, and we saw in the face the sudden transition from active danger to the mere exhaustion of a past conflict, even then the temperature chart gave no immediate indication of this critical change. The tracing of the eighth morning, when the patient was unconscious and like to die, was not appreciably different from that of the same evening, when he awoke from his first sleep, and it became certain that he would live. So fallible are set rules and directions, whether for the prognosis or treatment of pneumonia, upon the showing of a few of its symptoms.

What is the material afforded by this case, which is no exceptional one, for drawing conclusions as to the efficacy of treatment? Suppose this man had been bled at the time when bleeding was proposed, how admirable that procedure would have looked in relation to what immediately followed. And similarly of the alcohol: it would be easy to enlarge on its timely administration and marked effect, even to the saving of life; yet if the contention were raised that the sudden amendment that ensued was nothing else than the natural crisis of the disease happening at its usual time, it would be hard to deny that this was so.

It must be frankly admitted, then, that the very nature of pneumonia precludes the possibility of determining with certainty the effect of treatment in reducing its gross mortality. But of its effect in reference to individual symptoms there is not the same doubt. By careful observation it is quite possible to ascertain how these are affected respectively by particular methods designed for their relief. In other words, the treatment of pneumonia is the treatment of its symptoms. It is the mode of placing the patient in the best conditions for enduring a disease whose pains, duration, and special dangers are approximately foreseen, in the belief that by so doing the chances of recovery, be they more or less, are sensibly increased.

In an earlier part of this work we have indicated the general grounds on which prognosis is based, but with the addition that pneumonia does not always declare itself at the first as belonging to this or that category. In all cases the serious nature of the illness should be at once recognised and announced to the patient's friends, and all needful direction and precaution be given with a view to its contingencies.

It is as a general rather than a local affection that we set about the treatment of pneumonia. So soon as the diagnosis is determined, the condition of the patient becomes the first concern, the progress of the lung inflammation the second. To give to the body the largest possible amount of rest is the ruling principle of treatment. Its details are to relieve pain, to secure sleep, to feed, to control pyrexia, and, when necessary, to stimulate.

As regards *pain*, the common precept of authors enjoining 'rest in bed' is not of itself informing without some special directions as to the means by which rest is to be procured. We insist so often upon the spontaneous recovery of pneumonia, that we are apt to forget the distress that is suffered the while. First amongst its active troubles is the pain of pleuritic stitch, aggravated, more or less, by cough. Remedies are not powerless against either of these symptoms, yet they may remain unrelieved and a source of mischief in preventing sleep, because they are not complained of by the patient ; for, early in his disease, as we have seen, there creeps over him a certain lethargy so that his speech ceases to be an index of his suffering. But the catching pain with inspiration that we call stitch needs no verbal complaint ; it can be seen in the sudden arrest of each breath, and heard in the abrupt ending of the inspiratory murmur. Applications to the chest, whether hot or cold,[1] the former much aided by turpentine, help to control this pain. The employment of cold (long used in Germany, and now common in this country) has the extra advantage, as we shall presently show (p. 419), of reducing pyrexia. Such remedies, and perhaps fomentation most, get disparaged sometimes because ill-applied. The swathing of the chest in an ample fold of flannel, from which water quite hot has just been wrung, with diligent renewal at short intervals, so as to maintain for a full hour a continuous application of heat at a uniform and grateful temperature, is almost always serviceable. Yet the notion of a hot fomentation is often quite different from this. To foment with the least movement of the patient, and without leaving the bedclothes wet, needs practice and address. Unless done deliberately and with intelligence it is useless, or worse. Sometimes it so happens that a dry heat, as the application of flannel from the oven, or of a hot-water bag, is, as the patient is quick to perceive, of more use in removing pain than fomentation. Both wet and dry methods are valuable, and the more to be insisted on, because with some minds their simplicity is against them. The manner of performing these small offices, the skill and dexterity which can perform them efficiently, with no disturbance of the patient from what is being done, but only a sense on his part of benefit received without his co-operation, comprehends no small part of the total amount of service we can render to those who are undergoing pneumonia.

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[1] For treatment by the ice bag as a means of relieving pleural pain, see p. 421.

But the relief afforded by these appliances is apt to disappear so soon as they are removed. If, therefore, pleuritic pain form a prominent part of the patient's distress, fomentation may be supplemented by leeching. That the abstraction of blood from the neighbourhood of the pain should give more relief than its withdrawal from a vein in the arm is a fact not easily understood, but generally acknowledged. It is indeed the one feature of treatment which has lived through many changes of practice, and still survives unsupported by any theory with the high sanction of experience. Some special advantage may perhaps be gained by thus adding a new sensation at the very spot of the existing one. The leeches, three or four, should be applied as near to the pain as is consistent with their being also on the front of the chest, so that the patient may lie as best suits him during their application, and afterwards not rub the leech bites in moving. After leeching, hot fomentation may be reapplied as before, and at this particular juncture, if on account of pain sleep has been long absent, an opiate of moderate strength, or chloral, or sulphonal, operating with the soothing warmth and concurring with diminished pain,[1] may procure some hours of sleep which, without these preliminaries, a larger dose might fail to obtain. In the failure of the above measures subcutaneous injection of morphia *made over the seat of pain* may be applied, but it has seldom occurred to us to have resort to this measure for the symptoms in question.[2]

The very same means that succeed in allaying pleural pain will often benefit the cough as well. This latter symptom is seldom actively troublesome except at first, and as with bronchitis, a more copious and less viscid expectoration presently ensues, whereby this substernal pain and sensation of 'rawness' on coughing are removed.[3] With pain thus relieved we have next to await the further development of symptoms, careful lest the activity of the morbid process should prompt purposeless activity in drug giving, yet at the same time ready with special appliances should life be threatened by hyperpyrexia, severe dyspnœa, or extreme prostration.

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[1] Dr. R. E. Power, of Dartmoor, describes the following local treatment of acute pneumonia by turpentine :

First, a hot terebinthinate stupe is applied until the skin is well reddened ; then a little plain oil of turpentine sprinkled over the affected part ; finally, a blanket wrung out of boiling water covered with a dry blanket. 'I have had,' he says, 'patients delirious and gasping for breath, with sordes on the lips (patients who should have seen the doctor twelve hours previously), fall asleep as the last blanket was applied, and awake out of danger.' 'As a rule,' he adds, 'the active treatment need not be pursued very long.'

[2] In speaking of the relief of pleuritic stitch by the above measures, separate mention should be made of a rare form of pleurisy in connection with pneumonia, namely, the diaphragmatic. Heat and cold, leeching and hypodermic injection, will sometimes entirely fail to relieve in any degree whatever the intense pain of this affection. Lately a patient, a man aged twenty-four, was under the care of one of us, and died, after seven days' illness, of double pneumonia, presenting (as post-mortem examination showed) a striking example of this form of pleurisy. From his admission to within a few hours of his death he was in agony, shifting and turning from side to side to find some position of relief in a manner quite unusual with pneumonia. The application of ice was an entire failure, and hypodermic injection little better. The utter sleeplessness caused by the intense pain might well be regarded as determining the man's fate. Fortunately, the symptom in this shape is a rare one, and the statement above as to the relief of pleuritic stitch is generally true.

[3] Ease of cough and expectoration are sometimes promoted by the use

Yet while waiting for coming danger we may also anticipate it. Symptoms familiar in themselves may show in such degree as to suggest to the experienced observer early in the disease that its later stages will be of exceptional severity. There are certain indications in especial that give this warning ; complete anorexia, continuous insomnia, active delirium, prostration. Any one of these symptoms in prominence may serve as a signal of coming danger, while as yet the local condition gives no note of alarm. Want of appetite is, we know, the constant attendant of fever, but aversion to food, real inability to swallow or retain nourishment, such impossibility of feeding as makes ‘ restorative treatment ’ only a name, is a very serious symptom in view of the exhaustion of a week's pyrexia. Anorexia of this degree is not uncommon in pneumonia. No doubt it was aggravated in old days by nauseous drugs and a dietary of ‘ slops,’ not stimulating to appetite even in health. So far as is consistent with a nutritious fluid diet the patient's known tastes and habits are to be consulted. Customary meal times, a proper interval between the times of taking nourishment, some variety in food, all this must be respected as far as possible. It is true, of course, that both the material of food and the times for giving it need modification, yet what is ungrateful to the well can hardly be gratifying to the sick. Even

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of the ‘ steam draught inhaler,’ the water of which may be impregnated with succus conii. But moistening the air of the room by means of a long straight-spouted steaming kettle (the ‘bronchitis kettle’ of the ironmongers) may serve the purpose. The use of expectorants and tinctures, as such, is to be avoided as interfering with the cardinal objects of treatment.

the heartiest appetite has its seasons, and would be cloyed by the constant sight and smell of food.

Not seldom at the beginning of an ordinary pneumonia thickly furred tongue, headache, constipation, nausea, perhaps bilious vomiting, with yellowish conjunctiva and sometimes actual jaundice—the symptoms, in fact, of what is known as a bilious attack—will concur with this total loss of appetite and explain it. In such case a mercurial, such as blue pill followed by senna draught or with colocynth, or in the case of children grey powder with the compound jalap powder, will be almost always of service. Together with this dilute hydrochloric or nitro-hydrochloric acid may be given with spirit of chloroform in infusion of gentian or other bitter.

While it is unnecessary at the outset to make alcohol a part of the treatment, yet with the view to promoting appetite it may be desirable to give some form of it, such as dry sherry or champagne in moderate and measured quantity, not as a stimulant otherwise than indirectly. With such help we may so far arouse appetite as to get nourishment taken sparingly. That must suffice. The good work may be undone in over-anxiety to feed. Digestion gets taxed beyond its power, and nausea or an attack of vomiting succeeds.[1] There are cases, indeed, where the stomach intolerance is for a while so great, that in spite of the urgent need of nutriment it is best to abstain for a little from all food whatever by the mouth, saving ice or iced water with some drops of brandy, supplementing this by rectal suppositories.

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[1] Where beef-tea is not tolerated by itself it may be digested with hydrochloric acid, after the following simple manner, resembling Liebig's, and which we have employed repeatedly with good result. Take a pound and a quarter of lean beef, chopped fine and free from fat or sinew; pour on it one pint of water containing forty grains of salt and thirty minims dilute hydrochloric acid. Let stand for two hours, stirring occasionally. Drain off all the liquid possible and slightly press the residue on a muslin strainer. Then add to the residue as much cold water as the liquid is short of a pint. Stir up and again strain off and press. Makes a pint. In the case of children raw meat juice is often of service and to be preferred to the above. (See Chap. XII, p. 251.)

Hardly less important than the food he takes is the *air the patient breathes and the temperature in which he lives.* It would seem but reasonable in these days of rapid and easy transit that at the very onset of pneumonia (weather and other circumstances permitting), the town or city patient should be conveyed to the pure air and quiet of the country. It can hardly be doubted that the benefit felt by those in health from change of this kind would be shared in some measure by the sick. Yet custom and fashion rule in these matters, and we must seek the same end by other means. In reference both to air and temperature the very same rules apply to pneumonia as to bronchitis, its close companion. Among the first needs of a disease that implies morbid irritability and sensitiveness on the part of the respiratory passages is the utmost purity of air and its careful regulation at one temperature, with more or less of moisture according to the changing conditions of cough and spitting. Of the means of securing effectual ventilation without draught this is not the place to speak. The subject forms part of Hygiene, and is discussed in works on that subject.[1] Of the precise temperature and the degree of moisture no rigid rule can be given. The season of year, the particular climate, and, within limits, the sensation of the patient, must be the guide. Important and often difficult as is this part of the treatment, the physician is never to forget how apt it is, owing to its apparent simplicity and importance, to be overlooked or neglected by the ignorant, especially during the night, when it most needs attention.

In the matter of *sleep*, it is far better, in our judgment, to obtain it by removing the pain or the prostration that stands in its way than by dulling the senses with narcotics. These we believe to be mostly out of place in the earlier stage of pneumonia. To seek to abolish pain by large doses of opium or chloral, or by hypodermic injection, is to prefer ease to safety. That amount of narcotism that, in spite of pleurisy and cough, will so far blunt the sense as to admit of prolonged sleep, is not unattended with suffering of its own. Such sleep is unrefreshing, and the waking from it is often the waking to an aggravated discomfort and dyspnœa, owing partly to the accumulated secretion within the lung. The early resort to narcotics, like the early resort to alcohol, itself a narcotic, is an evil provision for the future needs of the patient.

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[1] See Parkes' 'Hygiene,' 6th ed. p. 147 *et seq.*

It is not only pain that keeps patients wakeful ; mere weakness may forbid sleep, and suggest a remedy which is indirectly sedative, namely food. The aids and the difficulties to feeding have been already alluded to, but whether more or less in any given instance, repeated dosing with morphia or opium cannot but weaken the digestive process. In the failure of indirect attempts to procure sleep by soothing pain and supplying adequate nourishment—measures on whose importance we cannot too much insist—it is best, we think, subject to certain exceptions to be presently stated, in the case of utter and obstinate sleeplessness, lasting over two or three days—a condition seldom without some delirium—to give one full dose of morphia by subcutaneous injection or by the mouth (according to age, &c.), repeating this after an interval of two or three hours, rather than to trust to uncertain narcotics like chloral or the bromides or sulphonal, or to repeated small doses of morphia, which are sometimes the reverse of narcotising.

Except for the relief of pain, any local application whatever to the surface of the chest, with a view to influencing the course of the pulmonary inflammation within the thorax (the application of cold to the surface, to be presently described, being laid out of the case), is, in our opinion, absolutely futile. It happens not seldom in the later stages of the affection that inspiratory and other pain is complained of, and for this mustard, or fomentation, or the ice bag, even leeching, may be called for as at the outset, but on no other account do we believe that these applications are of any service whatever.

The pneumonic fever, like other pyrexia in adults, is usually accompanied by constipation ; sometimes, however, the early days of the disease, and sometimes its crisis, are marked by the presence of diarrhœa. This may arise from accidental causes, such as alteration of diet, the copious draughts of fluid taken to allay thirst, or the too frequent administration of food. Sometimes it is the direct result of domestic medicine taken at the outset with a view to relieve the first feeling of illness, and which, from a prevailing instinct, is almost always aperient. Diarrhœa of such origin is short lived, and needs no treatment, while in the case of adults it is uncommon for this symptom, however originating, to be so persistent as to give trouble. Grisolle's observation of nine cases of profuse diarrhœa out of fifty-one patients suffices to show that it is exceptional. Occurring early, it is rather a source of discomfort than of danger, seldom needing the employment of drugs ; occurring late, it is sometimes among the symptoms of crisis, and disappears spontaneously. Careful regulation of diet, warmth to the abdomen, with a restricted use of such fluid as is not food, will commonly suffice to allay it. If not, laudanum in combination with some bitter astringent, such as catechu, or the aromatic mixture of chalk with opium, bismuth, or the compound kino powder may be given. It may be that opiates are already in use for some of the incidental symptoms already noticed. In such event the concurrence or persistence of diarrhœa may need enemata of starch with laudanum.

Diarrhœa continuing without abatement in face of remedies will lead to the suspicion of commencing ulceration of the ileum, and this in its turn to the possibility of the pneumonia having a tubercular origin, a rare event as we have shown, and one which the history and progress of the case will presently verify or disprove. In addition to the diarrhœa of crisis, there is that which sometimes attends the later stages of fatal pneumonia, and lasts till death, yet with no intestinal lesion to account for it.

A different account has to be given of this symptom in the case of children. But though diarrhœa is quite common at the outset of a child's pneumonia, it is not often either persistent or dangerous. It may be suffered for a while, and should it become necessary a few doses of the compound chalk and opium powder, combined with an equal quantity of Dover's powder (a grain or two of each, more or less, according to the age of the child) will probably suffice with appropriate diet to keep it in check. (See Chap. XII, p. 251.)

So far, in accordance with the principles stated at the outset, we have restricted our notice of drugs to those that are needed to meet special symptoms, and have ventured to express doubt as to the existence of any medical material whatever that can be described as good for pneumonia in the sense of a specific. But in respect for ancient usage and the survival through many revolutions of practice of a medicine that is at the least harmless, we may be allowed to retain the familiar saline draught. 'Neutral salines,' says Dr. Wilson Fox, 'favour the action of the skin, and thus reduce the discomfort from the pyrexia, and probably aid in the elimination of effete matters by the urine;' valuable properties, which need not be too strictly inquired into, inasmuch as the absence of all specific directions as to the quantity or mode of administration of this remedy tells pretty plainly in what light estimation it is in fact held.

Thus tended, the large majority of persons attacked with pneumonia in these days make a good recovery. In from three to eight days the fever slackens, or suddenly disappears, and the convalescence which shortly follows proceeds commonly without interruption until it ends in health. We have now to consider the case of those who are less fortunate, and to describe the treatment we believe to be the most efficacious in dealing with symptoms of an exceptional sort or of exceptional severity.

CHAPTER XXI

TREATMENT OF SOME PARTICULAR SYMPTOMS

Nervous prostration—High fever—Application of cold—Antipyretic drugs—Acute delirium—Use of alcohol; of narcotics—Asthenia—Extreme dyspnœa—Question of bleeding—Stimulation of external surface—Local treatment—Pleural effusion and empyema—Digitalis—Repeated stimulants—General results of treatment—Conclusion.

It is not always possible at the outset, as has been repeatedly said, to distinguish between patients that will do well and those that will do ill. Sometimes untoward symptoms at the beginning, such as sleeplessness and inability to take food, with or without great frequency of pulse and severe dyspnœa, make it obvious that the illness is likely to be severe, and the issue doubtful. The local as well as the general signs may give note of danger. The lung inflammation may be extensive, or it may involve both sides, or be conjoined with pericarditis or with empyema; or, commencing in a small area, it may rapidly spread so as to involve the greater part of the respiratory space. And apart from physical signs, there may be that in the patient's aspect which bodes ill. Of bad augury always is that condition which makes the aspect of pneumonia sometimes so similar to that of typhus or enteric fever—listlessness and indifference, with blunted perception and some mental wandering, a form of the disease, as has been said, somewhat more common with inflammation of the upper than of the lower region of lung. If with such symptoms the patient is known to have been of drunken habits, or still more, if with alcoholic excess there has concurred want of food, the speedy spoiling of the lung and consequent death may be regarded as probable. On the other hand, a degree of drowsiness and indifference (of the former especially in the case of children) is among the proper symptoms of pneumonia, and with a temperate history and fair capacity in taking nourishment need not excite alarm.

Pneumonia may threaten life by the *intensity of its pyrexia*. We have quoted examples to show that the degree of fever is not always an accurate measure of the patient's danger. Nevertheless, a sustained temperature over 104° ought not to be suffered, it cannot but be harmful in itself, and, in our opinion, the only effectual mode of reducing it is by the application of cold to the surface. Hyperpyrexia often concurs, as examination presently shows, with a condition of lung absolutely irrecoverable, and the temperature is but the index of a rapid process of disorganisation that must be fatal anyhow. Treatment by bathing or sponging seems less applicable to pneumonia, therefore, than to other acute diseases where the structure changes are less ruinous, such as enteric fever[1] and acute rheumatism. But when there is hyperpyrexia without marked physical signs of lung destruction, this method of treatment has sometimes proved successful.[2]

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[1] Liebermeister, in commending antipyretic treatment by bathing as especially applicable to enteric and typhus fever ('Handbook of Therapeutics,' vol. ii. p. 159, &c., translated by Dr. Matthew Hay), admits that less is to be expected from it in pneumonia where the local affection is of much greater importance. Nevertheless, this author states that even in pneumonia 'the treatment has yielded essentially better results than the usual expectant symptomatic treatment.' Thus, 'under indifferent treatment,' of 692 patients treated at Berne between 1839-67, 175 (or 25·3 per cent.) died; while of 230 treated 'since the introduction of antipyretic treatment' 38 (or 16·5 per cent.) died. It appears, however, that of this latter number all were not actually treated antipyretically. Reckoning those only in whom baths were used, we find 150 patients, with 16 deaths, a mortality of 10·5 per cent.

[2] Dr. Wilson Fox quotes three cases of hyperpyrexia, two of which recovered, one of these being pneumonia. In the first the temperature

The question of active antipyretic treatment in respect to pneumonia is not, however, limited to the exceptional occurrence of hyperpyrexia. The whole purpose of antipyresis, which has assumed so dominant a position in therapeutics, is to obviate the ill effects of the febrile process upon the organism. Hence it is of real value in such long continued fevers as typhoid, where these effects are more pronounced. In a brief, though not seldom severe type of pyrexia such as characterises pneumonia, it might seem that the need for such measures is less urgent. But as we have seen, there can be little doubt that the high bodily temperature, short as may be its duration, is responsible for certain grave complications in pneumonia, and especially for the symptoms of nervous disturbance indicated by insomnia, delirium, and prostration, to speak of no more serious effects. There would seem then to be rational grounds for recourse to antipyretic measures, and the problem is to determine in what way they may be best applied.

The most powerful, and in many respects the most useful, means for controlling pyrexia is that which has done such good service in the condition of hyperpyrexia, namely, the *application of cold.* There are many ways in which this may be effected, such as by immersion of the whole body in a bath at 80° for a period of 10 minutes, or in a bath of which the initial tempera-

rose to 110°, the patient (a woman of forty-nine, with acute rheumatism and pericarditis) being insensible and apparently in the act of dying. The pyrexia was reduced by cold water and the application of ice to the body. After rallying somewhat collapse was threatened, and heat was applied, the patient being *largely stimulated with brandy.* The bath was afterwards repeated at 64°, and subsequently ice bags applied to the spine, but these on the whole failed of their object. In the second case (that of a man of thirty-five), described as double pneumonia with pleural and pericardial effusion, the temperature rose to 107°. Scruple doses of quinine were given. After the first bath subsequent threatenings of hyperpyrexia were treated with wet sheet packing. This patient *took as much as thirty-three ounces of brandy in twenty-four hours.* A third case (of acute rheumatism), with a temperature of 108·9, died. (See 'Treatment of Hyperpyrexia by Means of the External Application of Cold,' by Dr. Wilson Fox, F.R.S.)

ture is higher, and which is gradually cooled down; but to effect the same result the immersion, in this latter case, must be longer. Or the body may be enveloped in a 'wet pack,' kept cold by ice, or invested with Leiter's tubes, through which a stream of iced water percolates; or sponging of the surface with cold or tepid water may be adopted; or only a portion of the surface may be constantly kept enveloped in a cold compress or in contact with an ice bag. It may be convenient to speak of these latter measures first, partly because they have been most favoured in pneumonia, and partly because their application is more simple, and likely therefore to be more generally employed than the others.

The value of cold compresses applied to the chest on the affected side was pointed out by Prof. Niemeyer,[1] who claimed for them great superiority to the routine method of poultices and hot applications. We have for many years followed this plan, but not in all cases (it being unsuitable for example where concomitant bronchitis has necessitated the use of poulticing), and although in not a few instances it has been found impossible to pursue the cold applications continuously throughout the pyrexial period, yet in almost every one there has been an appreciable reduction of the temperature. There is one great and unavoidable objection to this measure, and that is the difficulty of keeping the patient at rest, and the necessity of his lying on the healthy side whilst the compresses are being applied. For in a few minutes after its application the compress is no longer *cold*, and it has therefore to be constantly renewed; thus interfering with sleep, and, indeed, often preventing the carrying out of the method in its entirety. The usual plan is to thoroughly wring out a cloth in ice-cold water and place it over the side of the chest, the bedding being, course, protected by a drawsheet. In spite of the frequent disturbance the compress is often well borne, and the patient willingly submits to the inconvenience on account of the relief the application gives to the discomfort caused by high fever. Nor need the presence of marked pleurisy be a contraindication, for the cold itself will often relieve the pain ; but if this be so severe as to require the application of leeches, the compress is better suited than the poultice to follow such application. The drawbacks of compresses are partly obviated by placing the patient on the non-affected side, and lightly covering the other side with linen, upon which from time to time small fragments of ice may be placed, the water running off into a trough made by a macintosh suitably arranged. By this device, which we have used in some cases, there is none of the continual disturbance of the patient which is produced by the frequent renewal of the compress.

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[1] 'Text Book of Practical Medicine,' 7th ed. (Engl. Tr. 1871), i. p. 183.

It is owing to Dr. D. B. Lees[1] that a still further improvement has been made in this mode of application of cold locally to the chest. He has employed for this purpose the ice bag, and his experience is most encouraging. It is true that he claims for the application of cold something more than a general antipyretic action. He suggests, and some of the cases he records (with one of which one of us was intimately concerned, and can vouch for the striking effect produced) show apparently that an incipient inflammation of the lung may be arrested by this simple measure. It is possible, too, that an extension of the inflammatory action may similarly be checked in the course of the disease ; but the natural progress to an abrupt termination in pneumonia renders it difficult to endorse this surmise. Still, in whatever way it be regarded, there can be no question from the evidence adduced by Dr. Lees[2] that patients so treated do well, and of his 18 cases there were 4 at least in whom a favourable issue seemed determined by the plan. None of the patients died, a result of course not conclusive, especially in view of the youth of the subjects. Similar testimony has recently been given by a Swedish physician, Flindt, who only lost 3 cases out of 106 treated 'by ice,' including 10 of double pneumonia ('Lancet,' August 10, 1889).

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[1] On the Treatment of Pneumonia by the Ice Bag, 'Lancet,' 1889, ii. p. 890.

[2] And also by Dr. Goodhart in the discussion at the Harveian Society that followed the reading of Dr. Lees' paper. Dr. Goodhart had carried

Dr. Lees divided his cases into six groups, as follows :

(*a*) Cases in which immediate final arrest of the pyrexia process followed the application of the ice bag.

(*b*) Cases in which immediate arrest of the pyrexial process followed the application of the ice bag, and a relapse followed its removal.

(*c*) Cases in which immediate arrest of the pyrexia followed the use of the ice bag, a relapse followed its removal, and a second fall occurred when the ice was applied.

(*d*) Cases in which rapid fall of temperature followed application of ice, but relapses (apparently due to implication of fresh portions of lung) occurred while the ice was being continuously applied.

(*e*) Cases in which no immediate arrest of the pneumonia followed the application of ice, but obvious relief to symptoms resulted.

(*f*) Cases in which no very obvious benefit resulted from the use of the ice bag.

The fact that in certain cases (those in class *d*) there was supposed to be fresh extension of the inflammation, would seem to show that the treatment cannot invariably control the local process, and we are disposed to believe that mostly the good effect is due to the general diminution of the pyrexia. We have had but limited experience of this plan, and as

out the plan in eighteen cases—in eight with excellent result ; as to abatement of pyrexia, and early convalescence.

the patients in whom we have tried it were young adults, we lay no stress on the fact that they all recovered. Some difficulty, it must be admitted, was often experienced in adjusting the ice bag. The following cases may serve as examples :

CASE 1.—F. S., twenty-one, draper's assistant, admitted on January 14, 1888, on sixth day of attack. T. 103·8, P. 120, R. 50. In front dulness and tubular breathing at right apex to level of fourth rib posteriorly, and in upper axilla similar signs of consolidation. Lividity, tremors, delirium, insomnia. Ice bag applied over right upper lobe.

Crisis occurred during the following morning (seventh day), and ice bag was discontinued, the temperature falling to 99·4, and after a rise to 100·4 that afternoon it fell in evening to 98°. It remained subnormal throughout the eighth day, but on the ninth there was a temporary rise to 101·6, after which the pyrexia completely subsided. The crisis was marked by an increase of the delirium, and for some days after the patient remained in a melancholic state. At date of discharge (February 11) there was deficient expansion of the right apex, weak breathing, slightly increased vocal resonance, but no dulness. (This case will be found in fuller detail in Chap. IX, p. 166, Case VI.)

CASE 2.—Emma K., thirteen, admitted on February 27, 1888, the third day of attack. There had been malaise for three weeks previously. Signs of consolidation of lower lobe. She was ill-nourished, not livid. Severe headache. Herpes on the chin. Tongue moist. (Temperature on admission not recorded in notes.) Ice bag applied to left base. This was followed by a fall in temperature, and was continued until the sixth day, when she complained of cold (T. 102·2 to 103·6). Poultices were now applied.

Crisis occurred on the evening of the seventh day, the temperature falling from 103° at 2 P.M. to 97° at 10 P.M. Dulness disappeared on the tenth day.

CASE 3.—Juliet G., seventeen, admitted March 2, 1888, on second day of attack. Well-nourished, dry lips, dyspnœa, headache. T. 103·2, P. 128, R. 44. Dulness and tubular breathing over region of apex of left lower lobe, crepitation over the base.

Ice bag applied over left infrascapular region. There was much headache and delirium. The dulness extended over the whole scapular region, and tubular breathing became developed here and in infraclavicular fossa.

On March 4 (sixth day) the ice bag was omitted. Its application had been followed by a fall in temperature to 101°, but with a subsequent rise in the evening of the 5th day to 104·2. There was much pleurisy, the pain of which was relieved by leeching. Poultices were then applied. The crisis occurred in the course of the eighth day, the temperature falling to 97·2, but this was followed by a temporary rise to 102° the same evening.

Diarrhœa and slight albuminuria occurred just after the crisis.

Physical signs had not wholly disappeared by March 16.

CASE 4.—Caroline B., twenty-two, admitted June 20, 1888, on eighth day of illness. T. 103·6, P. 160, R. 28. Well-nourished, flushed, pain in left side, cough, shortness of breath. Dulness and tubular breathing at left base.

Ice bag applied to left base.

Temperature fell to 99·6 the same evening, without concomitant improvement in signs or general state. Increase of pleuritic signs, and leeching prescribed on next day. The temperature rose that evening to 104°, and remained between 102·6 and 103·4 during the tenth day, but on morning of eleventh it fell to 98°. However, it again rose to 103·6, and it was not until the following morning that it again reached 99°, nor until the fourteenth day that it was subnormal.

Ice bag discontinued on the tenth day.

Another case in which the ice bag was applied has been already recorded (Chap. IX, Case XV).

The plan of iced compresses above mentioned was used in three cases, the application being generally interrupted when the temperature fell to 101°. One was a severe case in a man aged 31, admitted on March 29, 1889, the fourth day of the attack. The compresses were applied on admission and continued till April 1 (seventh day), but only when the temperature rose above 101°. The crisis took place on the ninth day. Another was the case of a lad aged 15, pneumonia of the right apex. In this case it was long before the physical signs cleared up, and there was no definite crisis. A third case, a man aged 21, admitted on the fourth day, right apex pneumonia, complicated by pleurisy with effusion. Here crisis occurred on the seventh day.

In no case, however, was the application continued quite up to the crisis, its discontinuance being mostly due to discomfort, rarely to any appearance of signs of collapse. The application of ice bags, with due precautions, including the selection of patients who are not debilitated or aged, has certainly advantages over the compress.

Of wet packing of the whole body (serviceable no doubt in other forms of hyperpyrexia) we have no experience in pneumonia. A method involving so much disturbance of the patient is one we should hesitate to employ.

We may refer, however, to three cases in which, at some part of the disease, immersion in a bath was had recourse to, and with marked benefit. One of these has been mentioned in an earlier chapter, the case of Lizzie C., where there was a suspicion of drain-poisoning (Chap. X, p. 196). The others may now be briefly related.

James M., fifteen years of age, was admitted into the Middlesex Hospital on May 25, 1882, being the second day of his illness, which commenced abruptly with shivering, sweating, and violent vomiting. On admission his temperature was 103°, P. 120, R. 30, and the only physical sign was scanty fine crepitation at the left base. He had no cough. Next day dulness was present over the lower half of the left back, tubular breathing at angle of scapula, and occasional fine râle. At 2 P.M. the temperature rose to 105·8, and he was placed in a bath of 80° F. for ten minutes; at 3.30 the temperature was 102·8, but at 4.45 it had risen to 104·8. The bath was therefore repeated, and an hour later T. 102°. But it again rose, and at 2 A.M. on the 27th (fourth day) it was 105·4. No bath was now given, and at 8 A.M. T. 104°, P. 120, R. 36. There was still no cough, but the physical signs of consolidation of the left lower lobe had become more marked. Some dulness and distant bronchial breathing had also appeared at the right base.

It should be said that after each bath cold compresses had been applied to the left side of the chest, and on this day these were replaced by a similar compress applied to the left arm, which was continued until the 30th.

In spite of these measures the range of pyrexia was fairly high, viz. 102·2 to 104·8. On the 28th (fifth day) he began to cough and had some scanty rusty expectoration ; R. 48, P. 120, T. 102·2 to 104°. Same signs at left back, and dulness at right base. During the next two days there was no obvious change, but on the 31st (eighth day) the temperature began to fall, and next day reached normal, the pulse falling to 96, and the respirations to 36. The cough had become more troublesome. The physical signs now commenced to clear up, but there was evidently a little effusion into both pleuræ. On his discharge there still remained a slight patch of dulness over the left back.

Whether the two baths given in this case had any material influence upon the course of the disease it is hard to say, but it must be remembered that cold applications were continued throughout the pyrexial period. It may be added that, although the fever was high and crisis did not occur until the eighth day, there was no anxiety on the score of the pulse, and in spite of both lungs being involved, no marked distress in breathing.

The other case was certainly benefited by bathing, which was pursued more diligently, although the patient was a young child.

William R., aged 5, was admitted into the Middlesex Hospital on December 19, 1882, having two days before been attacked with severe headache, vomiting and becoming rapidly delirious. He had also pain in the left side. On admission T. 103·4, P. 120, R. 48. Only some coarse râles were audible at both bases. Next day there was dulness at left base, together with predominance of râle on this side ; P. 150, R. 60. At 2 P.M. T. 104·8, and cold sponging ordered then, and repeated at 4 P.M., when T. 103·4. This did not produce much result, for at 5 P.M. T. 104·2. The child was placed in a bath at 85° F. for fifteen minutes, the effect of which was to reduce the temperature at 6.30 to 102·4, and to 101° at 11 P.M. After the bath the child slept well and had a good night. On December 21 (fifth day) the temperature rose to 103° at 6 A.M. when the body was sponged. At 10.30 it reached 104°, when a second bath was given. This again produced some sleep, but did not long control the pyrexia. There was now a troublesome cough, and high-pitched tubular breathing, with fine crepitation over left back. At 2.45 P.M., T. 104·8, a third bath given ; at 2 P.M. T. 101°, and child asleep ; 5 P.M. T. 104·2 ; fourth bath, the temperature at 6 P.M. being 101° and at midnight 102°. At 6 A.M. on the 22nd (sixth day) T. 104°, fifth bath, 7 A.M. T. 101·2. At 8 A.M. T. 104·5, and at 9 A.M. T. 104·2, sixth bath; 10 A.M. T. 100·8; 11 A.M. T. 102·2, P. 148, R. 64. Dulness over left back well marked, tubular breathing and broncophony. In the afternoon there was a gradual fall in temperature to 99° at 8 P.M. Next morning, however (seventh day), the temperature rose to 103·4 at 6 A.M., and sponging was applied; at 9 A.M. T. 105°, seventh bath; 11 A.M. T. 101°; 1 P.M. T. 103·8, sponging. After this the pyrexia abated, and at 9 P.M. T. 98·4, and although next day there was a recrudescence to 102° at 3 P.M. the temperature was once more normal. On the 25th (ninth day) the maximum was 98·8, the usual record being 97·6. The child now slept a great deal, and there was no further pyrexia. The physical signs of consolidation of the left lower lobe speedily disappeared.

It should be stated that after the first bath it was remarkable how well the child bore the immersions: that they contributed to its comfort, and preserved its strength by favouring sleep and abating pyrexia, there can be no question, but there was no ground for believing that the treatment had any influence upon the course of the pneumonia, or hastened the crisis.

Of the *drugs made use of as antipyretics*—quinia, aconite, digitalis, veratria—we have already spoken.[1] More recently salicylic acid, owing to its admitted service in acute rheumatism, has been largely employed in other pyrexia, including that of pneumonia. Liebermeister fixed the antipyretic dose of salicylate of soda at a quantity corresponding to about 75 grains of the acid, 30 grains being equivalent to about 15 of quinine, which drug it nearly resembles in its antipyretic effects. Salicylic acid, however, is more prompt in its action, and owing to its rapid absorption should be divided into small doses given hourly. More often than with quinine the fall of temperature is attended with profuse sweating, so that a special diaphoretic action may be attributed to the drug. It is added that with cardiac weakness salicylic acid acts unfavourably upon the heart, and that sometimes with the fall of temperature a certain degree of collapse takes place, a fact especially noticeable in reference to its suitability in pneumonia. Yet, according to the same authority, there is no fever which of itself contraindicates salicylic acid, and in pneumonia amongst other diseases its antipyretic action appears surer than that of quinine.

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[1] The modern practice as regards antipyretic drugs in pneumonia may be gathered from the 'Report of the Col. Invest. Committee.' In the treatment of 1,066 cases quinine is the favourite amongst such remedies, having been used in 143 cases; aconite comes next, with 125; digitalis and salicylic acid were used respectively 65 and 41 times; veratria was used 4 times; cinchonidine once; and tartrate of chinoline once ('C. I. C. Record,' p. 56). Had the report appeared a year or two later no doubt antipyrin would have figured in it.

Antipyrin, antifebrin, and other allied drugs, so widely popular in the last few years, are of too recent introduction for any deliberate statement as to their value. According to Van Moorden, their effect in pneumonia and erysipelas is slowly produced and not very lasting.[1] It will not be denied that the reputation of these remedies has suffered in the recent influenza epidemic, or that in addition to mere lapse of time, fatal to so many new remedies, this experience is partly responsible for the comparative neglect that has befallen them.

We have ourselves but little experience of these modern antipyretic drugs in the treatment of pneumonia. In a few cases we have prescribed antipyrin in 10-grain doses, to be repeated hourly so long as the temperature remained above 101°, the administration being commenced whenever it rose again to 103°. But we are bound to state that in no material degree did the drug seem to exert a favourable influence, and the fear of inducing cardiac depression often led to its withdrawal before it had produced the effect desired. Thus in one case it was given to the extent of 110 grains in divided doses, without lowering the temperature more than 2°, and it was then discontinued, the temperature rising again to 103°. In this case, one of right basic pneumonia, the crisis, which happened three days later, was certainly not promoted by the antipyretic. In another patient 30 grains were taken on the sixth day, when the temperature was between 103° and 104°, but this, given in three-hourly doses, did not reduce the temperature below 102·6 ; the crisis occurred on the seventh day.

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[1] 'Handbook of Therapeutics,' vol. ii. p. 492, note by translator, Dr. Burney Yeo.

We have given antifebrin (in doses of 2 to 4 grains) in similar manner, but with hardly more encouraging result. In our opinion these powerful drugs are of small service in pneumonic fever, and they are apt to be mischievous. We may here take the opportunity of remarking that the production of antipyresis by means of drugs is never altogether satisfactory, apart from the special risks which may attend their use. It may, we think, be said that there is less rapid and less lasting improvement [1] in the patient's condition during such treatment than there is after the cold bath. The latter acts, of course, mainly by the impression it exerts on the nervous system. The antipyretic drugs also act, it may be supposed, upon the nerve-centres. But in the one case the action appears to be more stimulant than in the other, whilst, paradoxical as it may appear, the patient who is treated by cold is almost invariably soothed and enjoys some refreshing sleep. No doubt this is due to the reduction of the fever, but further, when the fever recurs it seems to be better borne by patients who have been so treated than by those who have taken drugs such as antipyrin or antifebrin.

Active delirium if persisting needs alcoholic stimulation. We have already referred (pp. 47 and 162) to instances of the disease ushered in by symptoms of delirium, which presently disappear, and have shown as well that nervous disturbance is especially prominent in the pneumonia of children at its first stage. It would thence appear that this symptom does not always indicate danger or need separate treatment. Yet active delirium in adults is so far exceptional that it should always suggest a careful scrutiny of the grounds on which the diagnosis is based.

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[1] In a note ('Lancet,' 1889, i. p. 730) relating to a case of pneumonia (marked 'rusty sputa, fine crepitations, and high-pitched resonance') treated by antipyrin, with crisis on third day, Mr. Counsell, of Liss, asks : 'Can pneumonia be cut short by antipyrin ?' The same question is applicable to the ice bag and every other form of treatment, and the answer, we fear, must be in the negative.

Typhus and enteric fever, when exhibiting lung congestion, are often mistaken for pneumonia, and with the former, as we know, active delirium is common. Assured, however, as to the diagnosis, this symptom would next suggest a history of alcoholism, or, failing that, of kidney disease, where among other signs of uræmic poisoning delirium may be remarkably active. In rare instances, it is to be remembered, meningitis may concur with pneumonia and be masked by it (p. 188).

In dealing with watchful delirium, it should be the first care of the physician to see enforced certain special directions as to nursing—absolute quiet, dim and even light, timely administration of food and stimulant, and the constant presence of a face and voice familiar to the patient. The nurse should be told that the symptom in question is more likely to yield to soothing and feeding than to more formal treatment. Where with the delirium there is headache it is often easeful to apply to the short-cut scalp a compress wrung out of ice-cold water, a procedure better than ice itself or other more complicated applications. When this sleeplessness finds no relief the necessity for a full narcotic dose or hypodermic injection arises. But first, along with sedulous administration of whatever nourishment is best taken, an alcoholic stimulant should be given in moderate dose often repeated, and together with this some such stimulant as spirit of chloroform or sulphuric ether, with solution of acetate of morphia. The effect of these measures is to be judged not only by the sleep obtained, but still more by the mental condition and the rate and resistance of the pulse, while so soon as they have obtained their immediate object they are to be temporarily suspended, since a uniform continuance of such remedies proves much less efficacious than their timely employment now and again as circumstances arise. Never should direct narcotics (amongst which alcohol in large doses has to be reckoned) be given without clear necessity, and the conviction at the time of their being in themselves evil.

Similarly in the pneumonia of drunkards, when there is sleeplessness and the delirium takes the busy form suggestive of delirium tremens (whether or not that term is strictly applicable), narcotics in full dose, whether hypodermic or other, together with free stimulation, is in our opinion as safe and as imperatively called for as with delirium tremens alone.

The near connection between delirium [1] and asthenia being understood, and the frequent occurrence of sleeplessness with both, it is still necessary to analyse these symptoms severally in order to determine the direction of treatment. Sometimes asthenia is of itself the threatening symptom, a fever-like prostration from which, as in typhus or enteric fever, recovery is still possible even in the last extremity. For this condition stimulation is the main treatment, and alcohol must be given freely,[1] yet with the one restriction, that it is to be suspended

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[1] Dr. Wilson Fox seeks to discriminate two conditions under which delirium occurs—that of weakness and that of blood-poisoning; but he admits that 'these are combined in various degrees,' and would prefer to be guided by general indications of asthenia (especially the low, muttering character of delirium) than by any theoretical reasoning respecting the origin of the symptom. (Reynolds' 'System,' vol. ii. p. 703.)

[2] See Dr. Wilson Fox (p. 705 *loc. cit.*), who relates having given thirty-six ounces of brandy daily for several days. See also case quoted in Chapter XX, p. 404.

It would be out of place to discuss here the action of alcohol. Its use as a stimulant rests mainly on empirical grounds; yet there is no department of practice in regard to which there is such general agreement. Of so soon as narcotism appears. Large quantities of brandy may be given in this extremity without producing any symptoms of alcoholic intoxication.

If, however, with great prostration there is much delirium ; if, notwithstanding occasional accessions of strength which the patient uses, under some delusion, to escape from bed or restraint, there is cyanosis, sweating, shallow panting breathing, with soft compressible pulse, then both opium and whatever else tends to deaden the senses to external impressions must be peremptorily forbidden. The time has passed for narcotics, whilst alcoholic stimulation must be used with caution and watchfulness lest it favour narcotism.

the physiological effect of alcohol the best known observations in this country are probably those of Professor Parkes ('Proc. Roy. Soc.' May 19, 1870), and these are mainly negative. Temperature was neither raised nor depressed, and elimination of nitrogen was not altered to any material degree. 'Two fluid ounces of absolute alcohol,' says Dr. Parkes, 'given daily to an adult seemed to increase the appetite ; with larger quantities it lessened or destroyed it. *The commencement of narcotism marks the time when appetite and circulation begin to be damaged.*' Upon this showing the effect and the dose of alcohol are matters for common observation rather than scientific rules. So also is the after effect of free alcoholic stimulation. In an affection like pneumonia, the later its use the more likely that the reaction following it shall not occur until the crisis has passed.

A short time before his lamented death Dr. Anstie, late physician to the Westminster Hospital, published some observations on the effect of alcohol in pneumonia ('Lancet,' March 21, 1874). In a case of double pneumonia six ounces of brandy were given daily, with the result that while temperature fell urea discharge was remarkably reduced. In a second case, with equable temperature, the urea fell on the first day of the alcohol stimulant, the fall continuing during the three succeeding days. Further evidence was in Dr. Anstie's possession (which, had he lived, he would have published) in support of his position, that 'in any pyrexial disease in which high temperature is conjoined with a large urea discharge, that conjunction is the strongest *primâ facie* reason for the administration of alcohol, and that a reduction of heat and urea discharge is to be regarded as evidence that the stimulation has been rightly adopted.'

The indications in respect of alcohol furnished by the older writers, as by Dr. Graves, namely, that it 'does harm if under its use the tongue becomes dry and baked, the pulse quicker, the skin hot, and the breathing more hurried,' may seem self-evident. Yet no practical physician will deny that these are the points to be specially kept in view after ordering a stimulant so potent (yet so often given by way of routine) as alcohol.

The character of the dyspnœa has now to be considered in view of the propriety of blood-letting. This must be learnt not merely from the frequency of respirations, but from their manner. Pleuritic pain may render the breathing shallow, catching, and frequent. No inspiratory act is completed owing to the pain it gives, and the patient seeks to make up by pace for what is wanting in completeness. Such method of breathing, judged from its frequency alone, would certainly mislead. Dyspnœa of this sort, dyspnœa that is *felt*—that produces, indeed, so long as there is consciousness, a lively sense of distress—is better than dyspnœa of the same frequency which is not actively distressing, although it is obvious to the sight that it is not more efficient in blood aeration than the other.

Again, causes apart from the lungs—pregnancy, abdominal distension by flatus, fluid, or tumour; deformity of chest, cardiac dilatation, anæmia, uræmia, and other things—may any of them contribute to heighten the breathlessness. Nor is the nervous element to be overlooked. It is said that hysteria disappears with acute disease, yet hysterical women and 'nervous' persons of both sexes may suffer inordinately from dyspnœa with pneumonia of no great severity. On the whole, dyspnœa is to be judged by what it does in respect of the function of aeration, and not by the sensations it arouses.

It is upon such considerations that the treatment of this symptom should be based. If within the first forty-eight hours considerable dyspnœa (let us say between 40 and 50 respirations per minute) correspond with a large extent of lung occupation, and if the stethoscope indicate that consolidation is only commencing, while at the same time the patient is attentive to the sensation of breathlessness, not cyanosed, and able to take nourishment and to get snatches of sleep, then we believe, looking to the usual issue of such cases, that it is best to adopt no more than the palliative measures already indicated, waiting for the change that may be expected within a short period. If, however, the patient be not encountered till later, say on the third or fourth day, a large area of his lung being now solid, with the dyspnœa he has had from the beginning still extreme, some blueness of face, and at the same time violent cardiac action concurring with weak pulse (p. 44), then in our judgment it is prudent to adopt the only known means of relieving such dyspnœa, namely, bleeding. The immediate (although too often temporary) benefit got from timely blood-letting far outweighs any theoretical consideration of the possible harm to be done by it. In other words, when dyspnœa is the prominent symptom, so that from its presence mainly the patient is denied rest, and when the physical signs indicate that the affected lung is solid, or mostly solid, then in the knowledge that at least temporary relief is almost always brought in that way, and in the belief that it is in any case harmless, we bleed from the arm to eight or ten ounces or more, according to the relief given. Nor do we hesitate to repeat such bleeding when the benefit of the first operation has been marked at the time, yet not permanent. Here is no theory of reducing inflammation, no promise of arresting or strangling the disease, or even of relieving the right side of the heart; only a procedure for the gaining of time where a certain period has to be lived through, a measure of relief which, imperfect and brief as it is, may yet serve its turn.

But, however striking the rally that follows blood-letting when judiciously employed,[1] it must be admitted that the improvement is in most instances short lived. Yet at the least, it gives an interval during which stimulants and other remedies may be applied with better hope than before, while the lessened dyspnœa may render sleep possible. Alcohol, we repeat, must be given at this stage with great caution, for in the condition contemplated of marked cyanosis and prostration, a state of stupor deepening into hopeless coma may very rapidly supervene. In view of this danger it will be sometimes prudent to discontinue alcohol and employ ether and ammonia as stimulants, together with ample turpentine stupes around the chest, and mustard and linseed poultices to the inner sides of the thighs, or, in suitable cases, to place the legs up to the knees in hot mustard and water.

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[1] In the present day all special injunctions in regard to blood-letting are set aside, and the sole direction has reference to the number of ounces of blood to be withdrawn, as though this could be stated for all patients alike, and that this old rule of producing an immediate obvious effect by the bleeding had ceased to be true. We do not in the present day bleed *ad diliquium*, and in the stage of pneumonia we are describing it is obvious that the patient's condition forbids any large abstraction of blood. At the

Such treatment, prompted by the patient's active distress from dyspnœa and increasing cyanosis, frequently alleviates suffering for the while, and may probably enhance the chances of recovery. It is otherwise where the very same need of blood aeration exists in one who is insensible of his own suffering. Little can be said in favour of bleeding when extreme dyspnœa goes along with extreme prostration, and the patient is at once breathless and unconscious.

What is to be learnt from the progress of the physical signs within the lung during this period of suspense? Not a great deal. It has been already mentioned that the earliest signs of lung disorganisation are not discernible by auscultation (p. 63). And so in the case of many patients whom we vainly endeavour to save, it is only after death that we discover that during the latter days of illness the condition was, in fact, past hope, the lung being already ruined, and life rendered impossible. Nevertheless, physical examination teaches much as to the extent of

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same time, the amount of blood drawn nowadays is sometimes so small that no effect, good or ill, can be expected from it; while the operation is often delayed so long that it would probably be useless in any case.

lung inflammation and its concomitants. Thus, the daily enlarging area of consolidation ; the supervention of bronchitis or of pulmonary œdema ; the occurrence of pericarditis or of pleural or pericardial effusion ; the variations in the force of the heart's contractions—all these are observations of importance in explaining symptoms and suggesting treatment. The collection of fluid within the pleural cavity, for example, whether serous or purulent (empyema occurring by preference with alcoholic patients) may be so insidious that only physical examinations can detect it, yet the importance of its timely removal is too obvious to need being insisted on.

In the present chapter we have confined ourselves strictly to our own experience and practice. It may be true that such experience and practice are far too limited to cover the whole ground ; but, on the other hand, the common habit of authors to rehearse the merits of a whole catalogue of remedies, but few of which they are in the habit of using, is not only confusing to the reader, but it helps to keep alive obsolete drugs that have nothing but tradition to recommend them. The young practitioner is thus perplexed in his choice between a large number of medicinal agents, all of them highly and similarly extolled. He flies from one to the other, and is disappointed with them all in turn, for each is tried incompletely with only a half confidence, while the thoughts are employed with the next. Sometimes he forsakes them all in despair, and trusts solely to brandy ; but more often the patient's death leaves him to regret that he made use of so few, and never hit on the right one.

Yet there is one medicine which, for its well-known service in cases of cardiac failure, is so obviously suggested in the treatment of pneumonia that we cannot altogether pass it over, namely, *digitalis*. This drug, though theoretically recommended for this affection in many quarters, we do not find actually employed on any large scale or with any striking success (p. 396). For its proved efficacy in other diseases it might easily become, perhaps has become, one of the routine remedies for pneumonia, and in such use with an affection so commonly recovering would be secure of a good reputation. Dr. Wilson Fox, referring, like other writers, to drugs of which he has little personal knowledge, says of digitalis, as of veratria, that 'it is deserving of a further trial' (p. 704), 'judging from his experience of it in large doses in delirium tremens with extreme prostration and very rapid pulse.' For ourselves we desire to say that having used it frequently in pneumonia when in want of a cardiac stimulant where there was much dyspnœa, cyanosis, and other signs of auricular distension, we have not found digitalis efficacious in removing those symptoms.

In like manner a large number of reputed stimulants—musk, camphor, phosphorus, valerian, turpentine, assafœtida, and many more—chiefly quoted from the literature of typhus, and each with its own advocate—are reproduced by writers of to-day with the old descriptions appended, but with no personal endorsement of these extravagant encomiums.[1]

It has been represented throughout this work that the time of crisis is virtually the time of safety, and that with this event drugs and stimulants may cease, and a speedy recovery be looked for. Some authors, however, lay stress upon the prostration that may follow close upon crisis, especially in old people, and sometimes with fatal result. Such relapse we consider quite exceptional both in old and young; while admitting that in pneumonia *apparent* crisis, indicated mainly by temperature fall, may precede the gravest symptoms (p. 180), and that in old people it is not very rare to find that after surmounting an acute attack of illness, whether pneumonia or any other, serious and sometimes fatal prostration will sometimes succeed.

---

[1] 'The change following its use was so unforeseen, so astounding,' writes Bourralier in reference to oil of valerian, 'that I have often heard persons attending my visits pronounce the word "resurrection."' (See Murchison, *loc. cit.* p. 278.)

With such reservation little need be said of the treatment of convalescence. Pneumonia, considering its severity at the time, entails as a rule but little wasting, and leaves the patient pretty much as it found him. Yet with perfect recovery of health traces of the bygone illness may, in exceptional cases, long remain. Thus the affected side may continue dull to percussion owing to thickened pleura, or forced inspiration may for an unusual time occasion redux crepitation, or the respiration may remain harsh and coarse with prolonged expiratory murmur. Such conditions, provided no portion of lung remain unresolved, are not inconsistent with full recovery. Patients often pass out of sight at this period, so that it is not easy to give any general estimate as to the duration of such signs.

It is unnecessary to add that special symptoms, such as exceptional wasting, anæmia, or night perspirations, may call for the employment of cod-liver oil, iron, and other tonics, or suggest change of air, especially with children and dwellers in towns, but inasmuch as the natural course is towards recovery it is impossible to measure the aid that these afford.

Something remains to be said of the treatment of some *complications and sequelæ*. Under the former heading we may specify bronchitis, acute rheumatism, acute nephritis, and delirium tremens ; under the latter, hydrothorax, empyema, pulmonary abscess, and gangrene.

Bronchitis, as we have seen, is never wholly absent, and is not to be regarded as a complication unless extending beyond the locality of the pulmonary inflammation and implicating both lungs. Its significance then depends upon considerations of age and previous health, which have been already discussed ; but in treatment the very same methods, with certain obvious modifications, serve for both affections. Rest, equable temperature, and the several expedients that have been mentioned to procure sleep, ease cough, and lull pain, apply to bronchitis as well as to pneumonia, with the reservation that expectorants are to be used more sparingly when the two diseases concur than with bronchitis alone. When, in addition to general bronchitis, there is pulmonary emphysema (a point to be very carefully investigated in patients at or beyond middle life), the complication, as we have seen, is serious (p. 103). It is to be remembered, however, that a sudden aggravation of dyspnœa in such subjects may be due, not to the pneumonia, but to the so-called 'cardiac asthma' often associated with emphysema. In that case relief may be given by the use of one or other of the many drugs that serve in true nervous asthma, and the patient will probably know from his past experience which of these serves best. The ætherial tincture of lobelia is often of marked service in the cases contemplated. It need not be added that the patient is not to be left to his own discretion in this matter, or that such remedies for asthma as depend upon producing depression are altogether unsuitable in pneumonia.

The concurrence of *acute rheumatism* with pneumonia calls for special caution in the use of reputed anti-rheumatic drugs. Thus the generally accepted fact that cardiac weakness forbids the employment of the salicylates virtually excludes such remedy in a disease like pneumonia, which subjects the heart to extra strain. Even the treatment by large doses of alkali, in the belief that by this procedure the risk of cardiac inflammation is lessened, ceases to be justifiable, in our opinion, when rheumatism and pneumonia concur. Patients exhibiting this complication often recover as we have seen. They have commonly youth in their favour. But they recover best when not actively treated with remedies which, whether beneficial or not in acute rheumatic arthritis, are certainly injurious to the affection that accompanies it and more immediately threatens life. It is the lung and the heart condition that should mainly govern the treatment, while the joint pains, relieved by local applications, should be considered in the second place so far as drugs are concerned.

It is somewhat otherwise with the rare combination of pneumonia and *acute nephritis.* Here there is not the same conflict as to the action of a single remedy in a complicated disorder. It is true that opium with its derivatives needs cautious use in nephritis, while at the same time there are certain symptoms of pneumonia that undoubtedly call for it. Nevertheless the existence of acute recent nephritis along with pneumonia does not in our experience practically hamper the practitioner in his choice of remedies. The need of caution and close observation in the use of narcotics we have repeatedly insisted on. This assured, we believe that the general principles of treatment in pneumonia, including the use of opium, are not greatly modified by the co-existence of acute nephritis.

But while in the two associations of pneumonia just mentioned, namely, that with acute rheumatism and that with acute nephritis, it is the condition of the lung that claims the main attention, the same rule does not hold when pneumonia occurs in the course of *delirium tremens.* We have already incidentally referred to the treatment of this affection, and in this place need only emphasise our belief that in delirium tremens, with or without this accompaniment, the first consideration has reference to the danger of death by asthenia. So far therefore the very same means of strengthening the heart, of procuring sleep, of feeding and stimulation, that apply to pneumonia apply to the nervous disease as well. But the prolonged insomnia and active delirium of delirium tremens will sometimes suggest the need for a direct narcotic which the lung condition may contraindicate. The necessity for sleep, however, in such cases is paramount, and when other measures, already adverted to, fail to procure it, we are of opinion that opium should be employed boldly. It must however be borne in mind that the subjects of delirium tremens may also be suffering from renal degeneration, and hyoscyamine rather than opium would be preferred in that case. Dyspnœa, if extreme, may be another source of perplexity in the condition contemplated, inasmuch as the venesection that sometimes relieves that symptom in pneumonia is not likely to benefit delirium tremens. Yet the result of bleeding in patients of this sort is so discouraging that we are little tempted to make trial of it.

Hydrothorax and empyema are to be reckoned as sequelæ rather than complications of pneumonia. A limited pleural effusion, indeed, like a localised bronchitis, is neither one nor the other, but an intrinsic part of the disease. But in rare instances, chiefly with persons of phthisical tendency, *fluid will accumulate within the pleural sac* so rapidly as to cause distressing dyspnœa and render immediate aspiration necessary. In the case of large fluid accumulation some caution is necessary lest the shock of quick and complete withdrawal should cause collapse or syncope. It is better in our judgment (especially with debilitated and aged patients) to remove such fluid gradually and in separate portions by successive aspirations rather than to encounter this risk. The remark applies only in the circumstances mentioned, namely, to bulky effusions of two or more pints. It is not founded on theory, but experience. Not seldom it happens that after a partial removal of fluid the rest is rapidly absorbed, so that no second aspiration is needed.

The treatment of *empyema* by free drainage and resection of rib is now generally recognised, and we have given two illustrations of it (Chap. IX, p. 181). But where empyema occurs early in pneumonia—where it is a complication rather than a sequel, the question arises whether free drainage should be established at once or else aspiration be employed alone in the first instance, leaving the rest until the termination of the pneumonic fever. Against immediate free drainage there is to be urged the necessity which is thus set up for daily dressing, when movement of the patient may be injurious, and the period of waiting is likely to be very brief. Against aspiration alone as a preliminary there is perhaps yet stronger objection speaking generally. The relief afforded is commonly partial and short lived, and it sometimes fails altogether. The precise method of procedure must depend on the particular circumstances of the case. In our own opinion aspiration should always be performed in the first instance, and its results be noted in respect of the size of the cavity, the amount of pus withdrawn, and the relief afforded. If in these particulars the operation is not satisfactory, free drainage should be at once established. The danger of retained pus overweighs the inconvenience of surgical interference at this early stage.

For the rest, *pulmonary abscess*, apart from gangrene, is seldom discoverable during life, and if discovered is little amenable to treatment. The diagnosis of *gangrene* depends, as we have seen, entirely upon the peculiar factor imparted to the breath and sputa, often in anticipation of any physical signs to indicate it. Such symptoms suggest the use of antiseptic inhalations and placing the patient in an atmosphere impregnated with carbolic acid. By such means some forms of pulmonary gangrene have been successfully treated, but in the case supposed, that is when directly associated with acute lobar pneumonia, it is very rarely recovered from. The propriety and method of surgical interference in such cases hardly fall within the scope of this work. It is right, however, to bring to mind the fact that, in a few recorded cases, a gangrenous cavity in the lung has been successfully tapped and drained. Where the indications are those of a circumscribed lesion, and the long continuance of the fetid expectoration and associated hectic fever are undermining the strength, resort to such procedure seems to us justifiable. Theoretically the chances of complete recovery would seem to be greater in the case of gangrene ensuing upon acute disease than when it is associated with an indurated and bronchiectatic lung. On the other hand, the risk of the incision setting up a putrid pleurisy or pyo-pneumothorax is not small, since it is just in these acute cases that the pleural sac remains unobliterated by adhesions.[1]

Here then we may conclude what will appear, by comparison with the more robust therapeutics that the very name pneumonia still suggests, but a bald account of the treatment of a grave disease. It would not be difficult to be more explicit and more peremptory, for there is no scarcity of drugs, nor is timorous misgiving the characteristic of those that commend them. Our own design has been announced at the outset. It is founded on the belief that pneumonia from the therapeutist's point of view may be roughly divided into two classes. The first of these, and at the present time numerically by far the largest, is sure of recovery, yet owes much to the skill and resource of the physician in rendering a painful affection as tolerable as possible. In the other, the more severe yet the smaller class, the final issue in life or death depends largely—we cannot, of course, say how largely—on treatment, the treatment, that is to say, of certain common symptoms such as delirium, dyspnœa, and sleeplessness, of whose pernicious effect we are not more certain than of the validity of remedies in large measure to relieve them. It is true that to this second class belong a proportion of cases, now more and now less common, wherein the inflammation is so rapidly destructive as to be altogether beyond the reach of treatment. Yet there are few indeed that so appear until the eve of death, and of pneumonia, as of enteric fever or typhus, we may say almost literally, while there is life there is hope.

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[1] Reference may be made to lectures by Mr. R. J. Godlee on the Surgical Treatment of Pulmonary Cavities, 'Lancet,' 1887, vol. i. p. 457 *et seq.*; and to a paper already referred to, 'Brit. Med. Journal,' 1885, vol. ii. p. 427.

So we write in this year, 1890, reflecting, as we believe, the opinion of those whose experience is the largest. But the treatment of a disease like pneumonia, of which the mortality varies at different times, is likely to undergo corresponding vicissitudes. So long as the results obtained are on the whole favourable we represent nature as beneficent, and charge ourselves with the business of seconding her efforts. But in altered circumstances it would be easy to change the figure, and revert to the old notion that regards the operations of disease as those of an enemy who is always to be opposed, and may sometimes be out-manœuvred. With a high death rate the significance of expectancy in treatment would be changed. Appeal would be made once more to the curative influence of tartarised antimony and the proved efficacy of early blood-letting; the study of the natural history of pneumonia would convey a new lesson.

At present, as it happens, we are content with the adoption of means which have the advantage of obvious reasonableness, resting not on the shifting sand of to-day's therapeutics, but on broad principles of conduct universally recognised and understood.

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